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INTELLIGENCE AS A FACTOR IN THE INCIDENCE OF ASIAN INFLUENZA

H. W. SMITH, M.A., PH.D.
Psychologist
and
E. F. TERBLANCHE, M.B., CH.B.
Medical Officer, Alexandra Institution, Maitland, Cape

This study is concerned in a general way with the question of the correlation between degree of mental defect and disease, not merely for its intrinsic interest, but also for its practical bearing on hospital administration. In particular, we wished to determine whether differences in intelligence levels were associated with significant differences in the incidence of Asian influenza.

It is widely accepted that mental defectives are also physically defective. Hollingworth states, 'Mental deficiency is not a special defect of judgment or reflection. It is a systematic, general inferiority'.[1] An obvious corollary is that the greater the degree of mental defect, the greater the physiological defect. Other inferences are, first, that the physiological deficit involves a lowered resistance and an increased proneness to disease and, second, that the greater the intellectual defect, the poorer the resistance and the greater the susceptibility to disease. These statements look plausible; nearly all doctors in the field of mental defect will subscribe to them. So widely experienced and eminent a scientist as Sir Cyril Burt writes: 'As we pass from the normal to the backward, and from the backward to the mentally deficient, the incidence of physical defect and disease, no matter what its particular form, tends regularly to increase.'[2]

The correlation between intelligence and physical health has been extended upwards in the intelligence scale so as to include normal and superior people. Carmichael's conclusion, based on thorough research by many workers over a long period of time, is that 'gifted children are superior to the age norms in 34 anthropometric measurements . . . gifted children are generally above the average in physical health and strength'.[3] Writing about a large number of adults with superior intelligence who had 25 years previously been subjects in an extensive study of gifted children, Terman[4] concludes, 'The gifted group is probably at least equal or superior to the generality in respect of general health, height, weight and freedom from serious defects. This is not surprising in view of the fact that medical examinations and anthropometrical measurements had demonstrated their superiority in childhood'.

We are unable to compare the incidence of illness among mental defectives directly with that of the normal population, because we have no reliable comparable data; but in an institution for mental defectives we have a wide range of intelligence levels, each with sufficient subjects to enable us to observe and compare the incidence of any illness at each level. In the light of the foregoing it was reasonable to expect that the incidence of Asian influenza would steadily increase as we went down the intelligence scale, to reach its peak with low-grade idiots. Our aim was to observe and examine this phenomenon.

MATERIAL

The setting is the Alexandra Institution for Mental Defectives, one of 3 such homes for Europeans administered by the Health Department of the Union of South Africa. It is conducted on hospital lines, the staff being mainly medical and nursing. It consists of 12 wards or blocks, each with a sister and nurses in varying stages of training. There are 2 matrons, 3 medical officers and a psychologist. At the time of the epidemic there were 866 European male and female inmates on the register.

The age range of the inmates was 0–85 years, the median being 29, and the interquartile range 18½ to 43 years. There were on the register 301 idiots (IQ 0–24), 342 imbeciles (IQ 25–49) and 223 morons (IQ 50–70). The institution roll was checked, patients on leave during the critical period were deleted, IQs and chronological ages were recorded, and the inmates divided into 2 groups, affected (with influ-

enza) and unaffected. The maximum temperature and the number of days each spent in bed were recorded.

We cannot think of any obvious environmental factors which could have operated selectively in the spread of the illness either from ward to ward, or from patient to patient. The buildings, staff, domestic arrangements, general nursing care and supervision all conform to a common standard. All the inmates apparently had equal opportunities for infection. In every case the appropriate diagnosis was made by the doctor, and the usual records were kept of the progress of each patient.

RESULTS

Nevertheless the wards were unequally affected. One with a preponderance of idiots was not affected at all, and is omitted from our treatment of the data. The remaining 11 displayed considerable variation as revealed by the following percentages of affected persons per ward respectively: 23, 27, 29, 36, 38, 39, 40, 40, 42, 62, 76.

For the purpose of our enquiry there were 750 patients left in residence at the critical time. Of these, 309 (41·2%) had influenza. Our first step was to obtain the incidence for each of the major intelligence groups, idiot, imbecile and moron. The results are reflected in Table I. As anticipated, we find the highest relative incidence among idiots, and the lowest among morons. Imbeciles fall somewhere between the two. The chances are 97 in 100 that the observed difference between idiots and morons is statistically significant, and the result allows a reasonably strong presumption that idiots were more susceptible than morons. Similarly, the chances are 96 in 100 that they were also more susceptible than imbeciles. The reliability of a difference is a stringent one; we are not entitled to an absolutely unequivocal verdict, and there remains a slight possibility that the difference could be due to chance. At this stage, however, we could have ended this enquiry with the conclusion that we have found reasonably strong evidence in support of the general statement that the greater the degree of intellectual defect, the greater the susceptibility to disease.

TABLE I. INCIDENCE OF ASIAN INFLUENZA, 309 CASES

	No. in Group	*No. with Influenza*	%
Idiots	253	124	49·01
Imbeciles	316	121	38·29
Morons	181	64	35·36

In view of the fact that the chronological ages of the patients were available, we wished to find out whether age was perhaps a factor, and we accordingly divided all our cases into age-groups with intervals of 10 years. We obtained the incidence for each. The results are presented in Table II. It is very obvious that the younger the inmates, the greater their proneness to contract influenza. Whereas old people above 70 years of age were not affected, and only 27·3% in the 60–69 years group, 74% of the children under 10 years old were affected. Half the affected cases were 21 years 9 months and younger, as against 32 years 11 months for the unaffected. The regularity of the intervals between the successive percentages shows that age was a consistent and important factor in the incidence.

TABLE II. AGE AND INCIDENCE OF ASIAN INFLUENZA, 309 CASES

Age (Years)	*No. in Residence*	*No. with Influenza*	%
0– 9	35	26	74·3
10–19	159	101	63·5
20–29	167	74	44·3
30–39	157	52	33·1
40–49	120	31	25·8
50–59	70	16	22·9
60–69	33	9	27·3
70–85	9	0	0
Total	750	309	

We know, however, that, because of their higher mortality rate, idiots have a lower average age than persons with higher intelligence levels. The median age of all the idiots on the institution register is 20 years 7 months as against 32 years 2 months for imbeciles, and 37 years 10 months for morons. There is accordingly ground for suspecting that the correlation between incidence of influenza and intelligence level, as revealed in Table I may be attributable to the lower ages of the idiots. The observed correlation may even be wholly due to age and not at all to an intellectual factor, as Table I would lead us to think.

The next step, therefore was to construct a frequency table, in which the relative influence of both intelligence and age could be observed as in Table III. This is read as follows: in the 0–19 years age group there were 101 idiots in residence, of whom 70 or 69·3% were affected with influenza. The intelligence factor has vanished in a very striking manner. In the age-group 40–85 years, the relative incidence is the same for idiots, imbeciles and morons. The small observed differences have no statistical significance. The same remarks apply to the 20–39 years group. With regard to the 0–19 years group, there are differences between idiots, imbeciles and morons, which have a fairly high degree of significance. Thus, the chances are 95 in 100 that the difference between imbeciles and morons is significant; but this cannot be due to an intelligence factor *per se*, because the incidence is lowest in the middle, the imbecile group, and highest in the moron group, with idiots in between, an arrangement which would make no sense on the basis of an intellectual factor.

The age factor is shown in strong relief in Table III; under each intelligence group, the highest incidence is among the 0–19-years-old subjects, and the lowest among the 40–85-years-old. Age would account for some of the variance in the spread of Asian influenza from ward to ward, but it is not enough. We have to assume the operation of some additional factor or factors to account for the differences between wards, and in particular for the fact that one ward was not affected. It is just possible that such a factor would also account for the difference just noted between idiots, imbeciles and morons in the 0–19 years age group.

TABLE III. INFLUENCE OF AGE VERSUS INTELLIGENCE ON INCIDENCE

Age	*Idiots*			*Imbeciles*			*Morons*		
(yrs.)	*No.*	*No.*	%	*No.*	*No.*	%	*No.*	*No.*	%
0–19 ..	101	70	69·3	76	43	56·6	17	14	82·4
20–39 ..	114	45	39·5	128	49	38·3	82	32	39
40–85 ..	38	9	23·7	112	29	25·9	82	18	22

It remains to be briefly noted that a comparison of the groups in respect of maximum temperature produced no differences. The average for idiots was 100·6°F, which

coincides with that of high-grade inmates. With regard to the time spent in bed, the average for the idiots was 6·6 days and for morons 8·7 days. There were no significant complications.

DISCUSSION

It is widely held that idiots are more prone to disease of all kinds than higher-grade defectives, and the latter in turn more so than normal people. In a recent survey at the Alexandra Institution (to be published shortly) the occurrence of illness among mental defectives during a period of 30 months was studied. A total of 6,629 referrals to the doctor were analysed in order to determine, among other things, what differences were to be found in the incidence of disease among idiots, imbeciles and morons. One finding was that these categories were referred to the doctor proportionately equally frequently. No one group was more susceptible to illness in general than the other; but when we observed the incidence of each illness by itself, we sometimes found small differences, and sometimes none at all. While some diseases favoured idiots, others favoured morons. We found no general, infallible rule to justify any generalization. Apparently the incidence of each individual disease has to be treated on its own merits, and its tendencies noted. The differences which we encountered could all be linked up with chronological age and such extraneous differences as ways of living, occupations and personal habits. Asian influenza is a disease in point.

It is misleading to generalize that the greater the degree of defect, the greater the incidence of disease, no matter what its form. As we have seen, if the statistical analysis is pushed to its limit, what originally looks like an intelligence factor vanishes, to be replaced by an age factor. Very often this will prove to be the case; the correlation between incidence and intelligence is nearly always a spurious one.

SUMMARY

This study concerns itself with the frequently alleged correlation between degree of mental defect and the incidence of disease. The recent epidemic of Asian influenza is examined for what light it may shed on the question.

Significantly more idiots than higher-grade defectives were affected. This finding would at first sight appear to vindicate the widely held view that the greater the degree of mental defect the greater the proneness to all kinds of disease.

But the observed correlation between intelligence and the incidence of Asian influenza proves to be a spurious one, for it vanishes when chronological age is taken into account. Age is found to be a consistent and powerful factor in the incidence; the younger the subjects, the higher the incidence. The median age of idiots is considerably lower than that of imbeciles and morons, and so we also find that Asian influenza had its highest incidence among them; age is the common factor and it displaces intelligence entirely.

An unpublished survey is briefly referred to for its bearing on the problem. The conclusion in general terms is that intelligence *per se* would appear to be rarely if ever a factor in the incidence of disease among mental defectives.

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REFERENCES

1. Hollingworth, H. L. (1931): *Abnormal Psychology*, p. 196. London: Methuen & Co.
2. Burt, C. (1937): *The Backward Child*, p. 168. London: University of London Press.
3. Carmichael, L. (1946): *Manual of Child Psychology*, p. 901. New York: John Wiley & Sons.
4. Terman, L. M. (1925): *Genetic Studies of Genius, I. Mental and Physical Traits of a Thousand Gifted Children*, p. 98. London: Oxford University Press.

BOOKS RECEIVED : BOEKE ONTVANG

Aortography. Its application in urological and some other conditions. By W. Barr Stirling, Ch.M., F.R.C.S. (Ed.), F.R.F.P.S.G. Pp. vii + 291. Fig. 155. 50s. net + 2s. 1d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

The Sick African. A Clinical Study. By Michael Gelfand, O.B.E., M.D. (Cape Town), F.R.C.P., D.P.H., D.M.R. Pp. 866. Fig. 164. 77s. 6d. Cape Town and Johannesburg: Juta & Co., Ltd. 1957.

Inhalation Analgesia in Childbirth. By E. H. Seward, M.A., D.M. (Oxon.), F.F.A.R.C.S., D.Obst. R.C.O.G. and R. Bryce-Smith, M.A., D.M. (Oxon.), F.F.A.R.C.S. Pp. viii + 58. Figures 11. 7s. 6d. Oxford: Blackwell Scientific Publications. 1957.

Medical Jurisprudence and Texicology. Tenth Edition. By John Glaister, J.P., D.Sc., M.D., F.R.S.E., F.R.F.P.S. (Glasg.), in collaboration with Edgar Rentoul, M.B.E., M.A., LL.B., M.B., Ch.B. Pp. xi + 720. 225 Illustrations, 72 in colour. 47s. 6d. net + 1s. 7d. Edinburgh: E. & S. Livingstone Ltd. 1957.

Current Medical Research. A reprint of the articles in the Report of the Medical Research Council for the year 1955-56. Pp. iii + 56. 2s. 6d. London: Her Majesty's Stationery Office. 1957.

The Malabsorption Syndrome. Editor David Adlersberg, M.D. Pp. iii + 252. Illustrations. $5.50. New York and London: Grune & Stratton, Inc. 1957.

Year Book of Dermatology and Syphilology—1956-1957 Series. By Rudolf L. Baer, M.D. and Victor H. Witten, M.D. Pp. 464. Figs. 62. $7.00. Chicago: The Year Book Publishers Inc. 1957.

Action des Radiations Ionisantes sur L'Organisme. Par A. Lacassagne et G. Gricouroff. Pp. 198. 17 Figures. 1.500 fr. Paris: Masson et Cie. 1956.

Electrocardiographie Endo-Cavitaire. Par H. Latour et P. Puech. Pp. ix + 294. Figs. 132. 3.200 fr. Paris: Masson et Cie. 1957.

Die Pathogenese Des Morbus Haemolyticus Neonatorum. Von Priv. Doz. Dr. Gerhard Martius. Pp. 70. DM 9.60. Stuttgart: George Thieme Verlag. 1956.

Heredo-Retinopathia Congenitalis. Monohybrida Recessiva Autosomalis. A Genetical-Statistical Study. By Carl Henry Alström in Clinical Collaboration with Olof Olson. P. 178. 9 Figures. Lund: Lund University. 1957.

Year Book of Cancer—1956-1957 *Series*. By R. L. Clark, Jr., B.S., M.D., M.Sc. (Surgery), D.Sc. (Hon.) and R. W. Cumley, B.A., M.A., Ph.D. Pp. 572. Figs. 210. $7.50. Chicago: Year Book Publishers, Inc. 1957.

La Balistocardiographie. Par G. R. Rager. Pp. 108. Figs. 32. 1,200 fr. Paris: Masson et Cie. 1957.

Érythrocytes et Erythropathies. Par P. Cazal, Professeur à la Faculté de Médecine de Montpellier. Directeur de l'Institut d' Hématologie. Pp. ix + 627. Figures 153. 5.000 fr. Paris: Masson et Cie. 1957.

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EDITORIAL

PRINCIPLES IN ANTIBIOTIC THERAPY

The antibiotics are still relatively new agents and although much has already been learned about their uses, and their capacity to do harm, it will take many years before their proper evaluation is complete. Certain basic principles are well established though not always observed in practice. It need hardly be mentioned that they should not be given just because a patient has a rise of body temperature. The identification of the infection and an estimation of its sensitivity to available antibiotics, based on bacteriological studies, might be regarded as ideal, but this scientific approach is not always feasible; moreover, delay may be dangerous for the patient and, further, it does not follow that a drug which is effective *in vitro* is necessarily also clinically effective. Another point to emphasize is that once the apparently appropriate agent has been selected initial doses must be adequate, so that if no response is obtained it can be inferred that the infection is not sensitive to the particular antibiotic administered.

Warnings have been given about the use of combinations of antibiotics. Such combinations might be regarded as desirable because some infections cannot be cured with one antibiotic alone, no matter how big the dose; or because organisms may emerge which are resistant to a particular antibiotic but respond to another; or because in some infections it appears better to give drugs before the causative organisms are determined; and, finally, the antibiotics might be combined to take advantage of their differences in toxicity. However, the results obtained with such combinations are complex, and while the effects may be additive or synergistic or sometimes merely due to the most potent constituent, there is the possibility that on occasion the actions may be antagonistic. Much depends on the size of the doses and the type of micro-organisms involved. Further work needs to be done on this important problem. At present it is not usually possible to state with certainty that particular pairs of drugs are antagonistic, but penicillin and aureomycin, and erythromycin and penicillin, and certain other combinations, have been shown to be antagonistic. Certain other combinations of drugs are known to produce synergistic effects.

With regard to chemoprophylaxis it is also necessary to go warily. In healthy persons prophylaxis has been success-

VAN DIE REDAKSIE

DIE BEGINSELS VAN ANTIBIOTIESE BEHANDELING

Die antibiotika is nog betreklik nuwe stowwe, en hoewel ons reeds veel omtrent hulle gebruike en hul moontlik beskadigende invloed geleer het, sal dit ons nog baie jare kos voordat ons hulle ten volle verstaan. Daar is sekere grondbeginsels wat reeds deeglik bevestig is, maar waarop daar in die praktyk nie altyd gelet word nie. Dit hoef skaars beklemtoon te word dat hulle nie somaar toegedien moet word omdat 'n pasiënt koorsig is nie. Die juiste uitkenning van die infeksie en 'n berekening van die besmetlike organisme se vatbaarheid vir beskikbare antibiotika, gebaseer op bakteriologiese navorsing, kan as die ideaal beskou word. Maar so 'n wetenskaplike benadering is nie altyd doenlik nie; die vertraging kan moontlik ernstige gevolge vir die pasiënt inhou, en 'n middel wat *in vitro* doeltreffend is, is nie noodwendig ook klinies doeltreffend nie. Dit moet ook benadruk word dat, sodra daar op 'n paslike middel besluit is, die aanvangsdosisse groot genoeg moet wees sodat, indien daar geen gunstige reaksie is nie, die geneesheer (sonder twyfel) tot die slotsom kan kom dat die infeksie bestand is teen die besondere antibiotiese middel wat toegedien is.

Daar is reeds gewaarsku teen die gebruik van kombinasies van antibiotika. Sulke samestellings kan soms as wenslik beskou word omdat sekere infeksies nie met 'n enkele middel alleen genees kan word nie—dit maak nie saak hoe groot die dosis is nie; of omdat sekere organismes na vore mag kom wat bestand is teen 'n gegewe middel maar wat vatbaar is vir 'n ander een; of omdat dit by sommige infeksies moontlik beter is om die middels toe te dien voor die veroorsakende organismes uitgeken is. Ten laaste kan die verskillende antibiotika saam gebruik word om voordeel te trek uit die verskille in hul toksiese hoedanighede. Die resultate wat met sulke samestellings behaal word, is egter baie kompleks, en hoewel die gevolge aan addisie, of aan samewerking, of soms bloot aan die aksie van die oorheersende faktor toegeskryf kan word, bestaan die moontlikheid dat die aksies soms met mekaar kan bots. Die grootte van die dosisse en die soort mikroörganisme betrokke by die siekte is ook baie belangrik en veel hang daarvan af. Hierdie belangrike kwessie verg verdere navorsing. Dit is vandag gewoonlik nog nie moontlik om te verklaar dat sekere pare middels mekaar sal teenwerk nie, maar dit is reeds bewys dat penisillien en oureomisien, en eritromisien en penisillien, asook 'n paar ander samestellings, nie saamwerk nie. Dit is bekend dat sekere ander samestellings van middels samewerkingseffekte as gevolg het.

Ook chemiese voorbehoeding moet baie versigtig toegepas word. By gesonde mense is suksesvolle voorbehoeding met penisillien behaal teen hemolitiese streptokokke-, gonokokke-

fully obtained with penicillin against haemolytic streptococcal, gonococcal and meningococcal infections. In sick persons with non-infectious conditions or with virus infections, chemoprophylaxis has not proved very successful in preventing bacterial infections. In rheumatic fever sulphadiazine and oral penicillin, and more recently aureomycin, have been advocated for daily administration throughout life to prevent recurrences. The wisdom of this is doubted by some physicians. Similarly in surgery there are objections to this form of prophylaxis against post-operative pulmonary and other infections.

In the chemotherapy of tuberculosis certain general principles are observed. Although drugs are very important they form only a part of the treatment of this disease, and the measures that were previously used are still important. This disease is not eradicated by a short course of treatment; foci of infection may light up and produce recurrence. No single drug is yet regarded as suitable by itself in the treatment, and in the great majority of cases, if not always, a combination of two drugs or even more is being used to prevent the emergence of resistant strains.

These few examples will indicate that the way to get the best results with antibiotics is beset with difficulties. However, in practice much good has been achieved by the intelligent and careful application of 'trial and error'.

The hazards involved in antibiotic therapy will be discussed in a future article.

Cornell Conferences on Therapy, 1953, **6**, 99. New York: Macmillan.
Robson, J. M. and Keele, C. A. (1956): *Recent Advances in Pharmacology*. London: Churchill.
Weinstein, L. (1955): Ann. Intern. Med., **43**, 287.

en meningokokke-infeksies. By siek mense met nie-besmetlike kondisies of met virus-infeksies, het dit geblyk dat chemoprofilakse nie juis daarin geslaag het om bakteriese besmettings te voorkom nie. Sulfadiazine en mondelikse penisillien, en onlangs ook oureomisien, is reeds aanbeveel vir daaglikse toediening by rumatiekkoors—die hele lewe lank—om te verhoed dat die siekte aanhoudend terugkeer. Sommige geneeshere twyfel of dit 'n wyse maatreël is. Ook by die chirurgie word besware geopper teen hierdie vorm van voorbehoeding van na-operatiewe longinfeksies en ander besmettings.

Sekere algemene beginsels word aangeneem by die chemiese behandeling van tuberkulose. Hoewel hierdie middels baie belangrik is, maak hulle maar slegs 'n deel van die behandeling van hierdie siekte uit, en die maatreëls wat voorheen toegepas is, is vandag nog belangrik. Hierdie siekte kan nie met 'n kort behandelingskursus oorwin word nie, want infeksiehaarde kan opvlam en 'n herhaling van die besmetting teweegbring. Vandag word nog geen een van die antibiotika, alleen en op sigself, as geskik vir behandeling beskou nie; by die meeste gevalle, indien nie altyd nie, word 'n kombinasie van twee middels of selfs nog meer as twee gebruik om die ontwikkeling van hardnekkige kiemsoorte te voorkom.

Hierdie paar voorbeelde onderstreep die feit dat die geneesheer met baie struikelblokke te kampe het voordat hy die beste resultate met die antibiotika behaal. In die praktyk is die intelligente en versigtige toepassing van die luk-of-raak metode tog al met heelwat sukses bekroon.

Die gevare van antibiotiese behandeling word in 'n toekomstige artikel bespreek.

Die Cornell-samespreking insake Behandeling, 1953, **6**, 99. Nu York: Macmillan.
Robson, J. M. en Keele, C. A. (1956): Recent Advances in Pharmacology. Londen: Churchill.
Weinstein, L. (1955): Ann. Intern. Med., **43**, 287.

TUBELESS GASTRIC ANALYSIS

Because it contains so many possible sources of error, the conventional fractional test meal is no longer looked upon as a reliable means of assessing gastric function. It is also now realized that, for practical clinical purposes, one rarely needs to know exactly how much acid is being secreted by the stomach. What is more useful is the knowledge whether the stomach is capable of producing hydrochloric acid at all. Both questions are answered by the augmented histamine test,[1] but it needs careful fluoroscopically controlled gastric intubation if the results are to be considered reliable.

Segal[2] devised a test for achlorhydria which does not require intubation. A carboxylic cation-exchange resin, in which the hydrogen cations are replaced by quininium ions, is given by mouth, together with caffeine to stimulate gastric secretion. In the presence of free HCl the resin gives up its quininium ions, which are absorbed in the intestine and excreted in the urine. If quinine is found in the urine after the administration of the resin, the stomach has secreted hydrochloric acid during the test. The test is not quantitative; there is no relationship between the amount of quinine in the urine and the amount of acid secreted by the stomach. Because the estimation of quinine is cumbersome, a new resin-preparation containing the dye azure A has been introduced;[3] hardly any effort is required to interpret the result.

If the test is positive, all is well, but false negatives may occur; one cannot conclude that a negative test means histamine-fast achlorhydria, which is what one usually wants to establish or exclude. Apparently attempts to combine the test with histamine as the secretogogue have failed. Malabsorption must often be differentiated from pernicious anaemia as a cause of megaloblastic anaemia, but under these circumstances the test is unreliable.[4] Poor renal function may also prevent the dye from appearing in the urine.

The limitations of tubeless gastric analysis are thus obvious, but it may be that they will be overcome by further refinements of the technique. It is a useful screening test under certain circumstances—but probably of less value than the estimation of serum pepsinogen.[5]

1. Kay, A. W. (1953): Brit. Med. J., **2**, 77.
2. Segal, H. L. (1951): Med. Clin. N. Amer., **35**, 393.
3. Segal, H. L., Miller, L. L. and Plumb, E. J. (1955): Gastroenterology, **28**, 402.
4. Bolt, R. J., Ossius, T. G. and Pollard, H. M. (1957): *Ibid.*, **32**, 34.
5. Spiro, H. M., Ryan, A. E. and Jones, C. M. (1956): *Ibid.*, **30**, 563.

IMMUNE GLOBULIN AND ITS USE IN PROPHYLAXIS AND THERAPY OF INFECTIOUS DISEASES

A. POLSON, D.Sc.

Senior Research Officer, C.S.I.R. Virus Research Unit, University of Cape Town

The results of physico-chemical studies on blood plasma have indicated that the material can be divided up into 5 families of proteins according to their mobilities in the electric field. These groups of proteins Tiselius termed according to their migration rates in decreasing order: albumin, alpha globulin, beta globulin, fibrinogen and gamma globulin. Gamma globulin is the serum fraction which harbours the bulk of the circulating antibodies, although lesser amounts may also be associated with a small component which has a migratory velocity between that of the beta and gamma fractions.

The gamma-globulin fraction, when separated from the rest of the serum proteins, is used to confer passive immunity in the recipient against a variety of diseases. The obvious advantage of this substance, which is derived from human serum, over hyper-immune globulin of animal origin is that undesirable factors such as allergic phenomena are eliminated entirely. The blood from which gamma globulin is separated is usually a pool derived from a large number of donors and for this reason it can be expected to have fairly constant antibody levels against diseases peculiar to the community from which it had been derived.

Gamma globulin is not a single protein entity like haemoglobin but contains a large number of components. On this account the practice adopted in the United States is to refer to it as immune-serum globulin (human). We hereinafter refer to it as immune globulin.

According to the United States Department of Health the minimum requirements for immune globulin are that it should contain $16 \cdot 5 \pm 1 \cdot 5$ g. per 100 c.c. of globulin having an electrophoretic mobility at pH 8·6 in diethylbarbiturate buffer not faster than $-2 \cdot 8 \times 10^{-5}$ c.m./volt/sec. and that the total protein should contain not less than 90% of such globulins.

Immune globulin can be prepared by a number of well-known methods such as ether fractionation,[1] ethanol fractionation,[2] or ammonium-sulphate fractionation. Of these methods the ammonium sulphate fractionation method is least satisfactory, since approximately only 60—75% of the material obtained can be regarded as immune globulin. Another important factor is that the virus of serum hepatitis, which may be present in the pooled serum, cannot be removed by this method.

Immune globulin is also prepared by a process which is allied to electrophoresis, namely, electrodecantation, in particular multi-membrane electrodecantation.[3] The method is briefly as follows: All proteins have iso-electric points. This means that at a particular degree of acidity, or pH, of the medium in which the protein is suspended the latter fails to migrate in the electric field. Below this pH the molecules move to the negative pole and above it they migrate to the positive pole. If the pH of the medium in which a mixture of proteins is suspended is adjusted to the iso-electric point of one of the components and a direct current is passed through the medium, the component which is at its iso-electric point will not migrate, but the associated fractions will move to the positive or negative poles, depending on whether they are above or below their iso-electric points. If a semi-permeable membrane is placed at right angles to the direction of migration of the molecules, they will migrate up to the membrane and form a concentrated layer there which will then, on account of its density, which is higher than the surrounding medium, sink to the bottom of the container, leaving the protein fraction, which is at its iso-electric point, suspended in the medium. The process of elimination of the migrating components is very slow in a container bounded by two membranes. If, however, a large number of membranes are introduced into the container at right angles to the direction of migration and separated from one another by suitable means, the process of separation can be speeded up greatly. If a series of such separation chambers, separated from one another by buffer solutions of the appropriate pH, is employed, the mixture of proteins can be passed in at the bottom of the first chamber and the partially purified component can be pushed over into the bottom of the next chamber, in which it can be further purified. In this way any desired degree of purity of the non-migrating fraction can be attained.

By this means, serum can be fractionated easily on a large scale into its main protein families, by making use of their differing iso-electric points. The latter are as follows:

Fraction	*pH iso-electric point*
Albumin	4·8
Alpha globulin	5·2
Beta globulin	5·4
Gamma globulin (immune globulin)	6·6–6·8

Immune globulin is separated at a pH of 6·6 but, on account of the fact that the gamma-globulin fraction contains the slowest migrating components in serum, it is possible by suitable control to obtain by this method a product containing components having differing iso-electric points in the gamma-globulin fraction and yet entirely free from the other faster-migrating serum fractions. The method is especially interesting in that the virus of infectious hepatitis, on the assumption that it behaves like other viruses which have iso-electric points below 6·0, is almost certainly eliminated.

USES OF IMMUNE SERUM GLOBULIN

The following conditions are listed as typical in which immune globulin can be of value, either as a therapeutic or as a prophylactic agent. Most of the clinical work on immune globulin has been carried out in the United States and has been published in American journals. Dosages in this review refer to material conforming to United States standards.

Agammaglobulinaemia

This condition is characterized by the presence of subnormal amounts or complete absence of gamma globulin in

the serum of patients. Persons suffering from this condition get repeated attacks of bacterial and virus infections and do not appear to develop any immunity on injection with bacterial vaccines as indicated by serological methods. The condition may be genetic or acquired. The sera of patients suffering from the condition show gamma-globulin levels of less than 75—100 mg. per 100 c.c. of serum. Normal human serum contains 700—1100 mg. of gamma globulin per 100 c.c. Confirmation of diagnosis of the condition by electrophoresis is essential. Paper electrophoresis is quite adequate.

In this condition treatment of recurrent infections with antibiotics has met with varying degrees of success, but more satisfactory results have been obtained by replacing the lacking antibodies through immune globulin therapy. This type of therapy for agammaglobulinaemia was first described by Bruton[4] and has been repeatedly verified by others. According to Bruton monthly injections of 0·3 c.c. of immune globulin per lb. of body-weight should be adequate to ward off recurrent infections. With this concentration Bruton succeeded in keeping his patients free of infection for an observation period of longer than 1 year.

Measles

The value of immune globulin as a prophylactic against measles has been shown repeatedly. Complete protection against the disease can be obtained provided it is applied within 6 days after exposure to infection[5-7] and provided adequate doses are injected. Evidence for the complete protection of children which were younger than 6 months and whose mothers were fully susceptible to measles has been supplied by Lundström.[8]

For the control of measles epidemics in institutions Lundström recommends the use of immune globulin which has been shown before to contain sufficient protective antibodies. He also recommends immune globulin as a prophylactic against measles for pregnant women to minimize the risk of possible abortions, premature births and foetal damage.

Of great interest is Bivings' finding[9] that, when an amount of immune globulin equivalent to 20% of that required for complete protection is given, measles is not prevented in exposed persons but a mild form of the disease can be induced with very frequent absence of the characteristic symptoms of exanthema. Greenberg *et al.*[10] concluded that immune globulin when given in inadequate amounts for complete protection reduces the incidence of measles encephalitis very significantly.

It must be emphasized that the course of the disease cannot be modified when immune globulin is administered during the actual disease. The explanation is that when the virus has invaded the susceptible cells, it is protected from the influence of the neutralizing antibodies in the immune serum.

TABLE I. RESULTS OBTAINED WITH IMMUNE GLOBULIN IN PROPHYLAXIS AND THERAPY OF INFECTIOUS DISEASES

Infectious disease	*Prophylactic*	*Therapeutic*	*Prophylactic dose (c.c.)*		*Remarks*
			Per lb. of body-weight	*For an adult of 160 lb. weight*	
Infectious hepatitis ..	Yes	No	0·02	3	The period of protection is exceptionally long.[13-15]
Measles	Yes	No	0·12	20	If given in amounts equivalent to 20% necessary for full protection a mild form of the disease can be produced. Administration of immune globulin is a safeguard against foetal damage when given early to pregnant women.
Poliomyelitis ..	Yes	No	0·2	30	Of value in the protection of polio contacts and pregnant women during polio epidemics. Possible period of protection 2-5 weeks.
Rubella	Yes	No	0·12	20	Convalescent serum has a definite prophylactic value. The same protective effect is obtained when immune globulin is administered in large doses. Very important for preventing foetal damage in pregnant women.
Varicella	Yes	No	Large doses		Large doses of the order of 50 c.c. for an average adult will probably have some value.
Pertussis	Yes	Yes	Hyper-immune human globulin (Cutter). Prophylactic dose for a child: 2·5 c.c. every 6th day. Therapeutic dose for a child: 2·5 c.c. every second day.		Very important for the protection of small children.
Homologous serum hepatitis.	Doubtful	Doubtful			Although the protection afforded is doubtful the Massuchusetts Dept. of Health suggests that dosages of 10 c.c. monthly over a period of 4 months will afford a degree of protection.[11]
Mumps, Orchitis ..	Yes	Yes	Large doses of immune globulin prepared from convalescent serum.		Ordinary immune globulin has no effect but convalescent immune globulin has a significant effect when given in large doses. It is very important in the treatment of pregnant women who have been mumps contacts to prevent possible foetal damage.[16]
Herpes simplex ..	Possible	Possible			Of beneficial value in treatment of herpes-simplex infections in children with eczema.[18]

However, it is a well-known fact that measles tends to reduce the bodily resistance against secondary invaders. Pneumonia and otitis media are frequent complications which follow on measles and although immune globulin has no effect on the course of the disease itself, it should have a definite prophylactic effect against the secondary invaders.[8]

Infectious Hepatitis

There is evidence that immune globulin contains large amounts of antibodies against the virus of infectious hepatitis and it is used with considerable success in preventing the disease in contacts.

It has been found that dosages of 0·02 c.c. of immune globulin per lb. of body-weight is sufficient to prevent the spread of the disease in military establishments and in children's homes and to protect laboratory workers in hospitals who handle infected material. The period of protection is exceptionally long.

Poliomyelitis

Immune globulin contains demonstrable amounts of antibodies against all three types of poliomyelitis strains. In tests carried out in the United States it was shown that it can be used prophylactically with considerable success. Unfortunately, however, the period of protection is not long.

The Massachusetts Department of Public Health recommends a prophylactic dose of 0·2 c.c. of immune globulin per lb. of body-weight.[11] This dosage produces significant protection from the 2nd through to the 5th week following injection.

Protection against poliomyelitis by means of immune globulin is of value in pregnant women and contacts during epidemics. It may also be used as an additional safeguard if tonsillectomy or other operations have to be performed on children when poliomyelitis is prevalent. Adequate protection in the majority of cases would probably be afforded by 10 ml.

The suggestion has been made that immune globulin may usefully be employed in conjunction with vaccination by means of attenuated (avirulent) live poliovirus, which it is hoped may give more solid results than the formalized vaccine. The object is to make certain that no harm will result should any virulence be present in the vaccine. A dose of 5 ml. of immune globulin should suffice for this purpose. This use of immune globulin has no adverse effect on the potency of the vaccine.[19] It is not anticipated that the live vaccine will come into extensive use in the immediate future; and it is not likely that immune globulin will be available on the large scale that its extensive use for this purpose would entail.

Rubella

A considerable amount of work has been done in Sweden[8] and Holland[12] during epidemics of rubella, and it has been shown that if whole serum taken from persons known to have had rubella recently is injected into contacts in doses of 25—50 c.c. then the protection afforded is 100%. If such sera are not available then it is recommended by the Massachusetts Department of Public Health that a dosage in adults of 20 c.c. of immune globulin be used. This dosage affords definite protection in pregnant women.[11]

In Table I further particulars are given of results obtained with immune globulin in the prophylaxis and therapy of infectious diseases.

CONTRA-INDICATIONS

No contra-indications for the use of immune globulin have ever been found. On account of the fact that it is of human origin, persons injected with immune globulin are not sensitized against it and therefore no precautions are necessary when repeated injections are administered. Also on account of these factors no harm can be caused through administering too large doses and when doubt exists as to the size of dose to inject it is better to err on the high side.

Further information on the clinical use of immune globulin can be obtained from an excellent review by Lundström.[8*]

REFERENCES

1. Kekwick, R. *et al.* (1946): Nature, Lond., **157,** 629.
2. Cohn, E. J. *et al.* (1950): J. Amer. Chem. Soc., **72,** 465.
3. Polson, A. (1953): Biochim. biophys. Acta, **11,** 315.
4. Bruton, O. C. *et al.* (1952): Amer. J. Dis. Child., **84,** 732.
5. Stokes, J. jr. *et al.* (1944): J. Clin. Invest., **23,** 531.
6. Janeway, C. A. (1948): *Ibid.*, **138,** 859.
7. Vahlquist, B. (1951): Svenska LäkT., **48,** 2152.
8. Lundström, R. (1955-56): Neue öst. Z. Kinderheilk., **1,** 197.
9. Bivings, L. (1953): J. Pediat., **43,** 401.
10. Greenberg, M. *et al.* (1955): *Ibid.*, **46,** 642.
11. Massachusetts Dept. of Health (1954): New Engl. J. Med., **250,** 417.
12. Lundström, R. (1952): Acta. paediat., **41,** 583.
13. Stokes, J. jr. *et al.* (1945): J. Amer. Med. Assoc., **127,** 144.
14. *Idem* (1951): *Ibid.*, **147,** 714.
15. Ashley, A. (1954): New Engl. J. Med., **250,** 1090.
16. Grönvall, H. and Selander, P. (1948): Nord. Med., **37,** 409.
17. Kaude, J. and Wehmeyer, A. (1955): Arztl. Praxis, **7,** 1.
18. Barrow, G. (1954): Brit. Med. J., **1,** 482.
19. Gand, S. Personal communication.

* Immune globulin in a 16% strength, separated by the method of electrodecantation, is prepared by the Seravac Laboratories, Cape Town, on behalf of the Western Province Blood Transfusion Service, and is obtainable from the Western Province Blood Transfusion Service. This material conforms to United States Department of Health standards. It has been found to be clinically pure, sterile and apyrogenic. Assay by Dr. James Gear, of the South African Poliomyelitis Research Laboratory, shows that titration of antibody of the three types of poliomyelitis virus gave the following results, type 1, 1 : 800; type 2, 1 : 3,200, type 3, 1 : 1,600.

THE THIRD WORLD CONGRESS OF CARDIOLOGY

The Third World Congress of Cardiology will be held in Brussels on 14-21 September 1958. The Secretary of the Congress has requested that 'participants residing in a country where there is a society of cardiology should send summaries of their communications to the secretary of their national society before 1 February'. It is therefore suggested that members of the Southern Africa Cardiac Society who wish to submit communications should transmit an abstract of not more than 200 words to the National Secretary (Dr. L. Vogelpoel, Cardiac Clinic, Groote Schuur Hospital, Observatory, Cape Town). Two copies are required, typed in double line spacing and they must be sent in before 1 February 1958.

A preliminary booklet giving information about the Congress has been sent to the secretary of each provincial branch of the Cardiac Society. Further information can be obtained from the Secretariat, Rue Mercelis 80, Brussels, Belgium.

THE GENESIS OF THE S-T SEGMENT AND T WAVE*

J. KELMAN DRUMMOND, M.B., CH.B., M.R.C.P. (EDIN.)

Durban

The interpretation of the electrocardiogram as attempted by various authors always proceeds simply and satisfactorily until it reaches the explanation of the S-T segment and T wave, the phase of repolarization, when it always becomes frankly presumptive and unsatisfactory. There does seem to be a simple and reasonable explanation of this phase of the electrocardiogram. Classical works attempting to explain this phase illustrate the depolarization and repolarization in terms of a single muscle cell, the latter phase being opposite in polarity to the former phase and the whole reaction proceeding in a simple fashion from the normal balanced ionic state to the unbalanced state and back to normal.

Depolarization may be termed a primarily electrical phase, the excitation wave proceeding through the myocardium and throwing the muscle cells into a state of ionic imbalance. In this way the QRS phase can be easily explained with, for example, the height of the R wave rising over the left ventricle when hypertrophied muscle has to be transversed by the vector, or splintering or widening of the QRS occurring when the block of a bundle, or ischaemic change elsewhere, causes the vector to pursue a more circuitous course relative to the exploring electrode.

Repolarization, however, depends primarily on biochemical change, for normal polarity will not be reached until the ionic balance is restored. As such it obviously takes longer than the first phase, for the S-T segment is longer than the QRS in duration and furthermore it really bears no true resemblance to the QRS, try as we may to make it fit. This occurs because of certain obvious conditions relating to the repolarization process:

Firstly, we are not dealing with a single cell but the entire myocardium and the order in which this is depolarized will affect the repolarization. Lepeschkin[1] points out that the subendocardial muscle is activated earlier but has a longer action potential than the subepicardial muscle and therefore the completion of depolarization, and thus the onset of repolarization will be fractionally delayed in the former.

Secondly, the order in which the myocardial cells are activated will affect repolarization too and, for example, with a bundle-branch block this order may be grossly disturbed.

Thirdly, the ionic state of the extracellular fluid has an important effect on repolarization, as in hypokalaemia, the transfer of ions across the cell membrane varying according to this ionic state with well-known electrographic sequelae (Tepeschkin[1]).

Fourthly, the blood supply to the individual cells and their nutritional state has an important effect on polarity, for a healthy cell when activated must be capable of generating far greater negative polarity than a poorly nourished one. The nutrition of an individual cell must be directly concerned with the efficiency and magnitude of transfer of the ions across the cell membrane, and these factors, acting both qualitatively and quantitatively, will inevitably influence the electrically 'discharged' cell in the direction of a greater or lesser negative polarity. It is probable that even with the simple process of aging, regardless of blood supply, the efficiency of this ionic exchange is likely to diminish.

* A paper read at a meeting of the Natal Section of the Southern African Cardiac Society, Durban, 1957.

S-T SEGMENT AND T WAVE

Bearing these factors in mind, the form of the S-T segment and T wave seems to follow sensibly. The normal S-T segment is iso-electric. At the completion of activation we have all the cells of the myocardium uniformly depolarized, with repolarization, at first, proceeding uniformly without. At this stage, then, since the change is uniform, an electrical bipole does not exist and this segment is iso-electric. Following this we have the normal upright T wave, which means that at this point an electric bipole has developed, with positive polarity relative to the praecordial electrode. If it is true that the subendocardial muscle is the last to commence repolarization, then it is likely that the subepicardial muscle will return to normal polarity in advance of the former, and the expected bipole then occurs, for there will be a residuum of depolarized and negative subendocardial cells.

Similar reasoning appears applicable to the abnormal S-T segment and T wave. It is not proposed here to discuss the gross displacement of the S-T segment associated with a recent infarct, for experimental work would seem to confirm that the 'injury current of rest' would account for this. Similarly the gross displacement of the S-T segment associated with a bundle-branch block appears to be due to the fact that subepicardial depolarization is still proceeding while the S-T segment is being inscribed. Of some interest is the fact that recent subepicardial injury produces the opposite effect to recent subendocardial injury, though again the current of injury accounts for the changes. But subacute or chronic ischaemic change, whether exaggerated by exercise or not, invariably produces an S-T depression, regardless apparently of the particular site of the ischaemic change (that is to say, when it produces any ECG change at all). Similarly, as the 'strain patterns' involve the S-T, change is in the direction of depression while the T tends to become inverted. These latter patterns demonstrate the existence of an electrical bipole which is negative relative to the praecordial electrode.

In regard to these changes it is important to recognize two features relative to ischaemic change, whether due simply to atheroma in the coronary artery or to left ventricular stress, as for example in systemic hypertension.

Firstly in terms of the pressure gradient, the more distal tissue supplied by an artery is likely to show greater ischaemic effects than the more proximal. In the myocardium this means that the endocardium is likely to suffer more severely than the epicardium and, by and large, the pathological changes confirm this. (Of some importance here is the fact that the S-T segment and T wave are inscribed during

mechanical systole, which is likely to accentuate these effects.)

Secondly, when the ischaemic effects are established for any length of time the progress of the activation wave from endocardium to epicardium is likely to be slower and repolarization in the latter is likely to be fractionally delayed in onset.

Bearing these points in mind let us examine the behaviour of the S-T segment when ischaemia occurs. The first change is commonly a little S-T depression. Since, now, the subepicardial muscle is better oxygenated than the subendocardial muscle, the better nutrition there will enable these cells to develop a greater negative polarity than the less well supplied subendocardial muscle. The result of this is that, while the whole myocardium is uniformly depolarized at the stage of the S-T segment, there will arise a weak electric bipole, negative relative to the praecordial electrode, and S-T depression is recorded. This change would obviously be accentuated by exercise and further S-T depression occur. Furthermore, since the S-T segment is being inscribed during mechanical systole, when blood flow is virtually halted, any ischaemic effects are likely to be progressively exaggerated and the S-T segment will then demonstrate the characteristic 'sagging' form of coronary insufficiency.

The next change recorded in the ECG is flattening and then inversion of the T wave. This change is related to the later commencement (and completion) of repolarization in the subepicardial muscle, due to the delay in the activation wave referred to above. At first, with an upright T wave, the endocardium is the last to remain negative. Then for a time subendocardial and subepicardial repolarization are synchronous and the T wave is likely to be flat and, finally, with more advance change, the subepicardium is the last to return to normal polarity and the negative T wave results.

Applying the same rules to a myocardial infarction, after the current of injury has disappeared and the S-T segment is once again iso-electric with the T-P interval, the question whether the T wave is going to remain inverted or return to the upright position will depend on the absence or presence of ischaemic change in the remaining functioning myocardium.

Applying the same rules to a pericardial effusion we find at first that normal subepicardial cells are subjected to pressure which would cause minor reversible ischaemic effects. This means that the subendocardial cells, unaffected at this stage, would be capable of generating greater negative polarity during the inscription of the S-T segment, and an electric bipole relatively positive to the praecordial electrode would develop. Thus we should obtain the elevated S-T segment and, since the myocardial cells at this stage are themselves healthy, the order of relative subendocardial depolarization and repolarization would not be affected and the T wave would remain upright. Later, however, the continuing tamponade would start having more severe and extensive ischaemic effects and, as S-T segment and T wave come down and are finally reversed in polarity, we are observing the same changes referred to previously as those of ischaemic change.

The same rules appear applicable to the effects of digitalis. Here we are dealing with an abnormal myocardium but the digitalis delivered by the blood stream will, by its acknowledged direct action on the myocardium, be more able to help the better supplied subepicardial muscle and a greater relative negativity there will cause depression of the S-T segment.

In this country we are familiar with bizarre displacements of the S-T segments and T waves as recorded by Grusen.[2] These changes may on occasions fluctuate from day to day or week to week or may be altered by exercise.

Gillman[3] has recorded, as part of a disturbance of nutrition, an abnormal uptake of iron and deposition of this iron in the myocardium (as well as in other organs). Part of this iron deposition, he believes, involves break-down of the cell cytochromes. Here apparently we have cellular change, reversible or not, involving the metabolism of the cell, and therefore ionic change must similarly be involved, and its site, distribution, and extent in the myocardium will determine the deviation in the S-T segment and T wave likely to be obtained. (These cells, as conductors of the activation wave, appear little affected, and the QRS changes, if they occur at all, are minimal.)

SUMMARY

There appears to be a logical explanation for the behaviour of the S-T segment and the T wave.

The S-T segment depends on the relative polarity of subendocardial to subepicardial muscle, while the T wave depends for its sine on which of these regions is the last to return to normal polarity.

The QRS portion of the electrocardiogram may be looked upon as the stage of electrical 'discharge', the cells acting with greater or lesser efficiency as simple electrical conductors.

The S-T segment and T wave, however, depend for their form on a primarily biochemical return to normal of the myocardial cell, and the form of this portion of the electrocardiogram is dependent on factors such as blood supply and cell nutrition to a far greater extent than the QRS portion.

1. Lepeschkin, E. (1957): Circulation, **15,** 78.
2. Grusen, H. (1954): *Ibid.*, **9,** 860.
3. Gillman, J. and Gillman, T. (1951): *Perspectives in Human Malnutrition*, p. 250. New York: Grune and Stratton.

RADIO-ACTIVE FALL-OUT AND ITS HAZARDS TO MAN

W. R. McMURRAY, M.Sc. (NATAL), D.PHIL. (OXON.)

Health Physicist to the South African National Physical Research Laboratory

PART I. FACTS AND FIGURES

Radio-active fall-out, as everyone knows, is a world-wide result of nuclear-weapon testing by America, the United Kingdom and Russia, and the amount and extent of this fall-out is directly related to the specific weapon-testing programmes of these three great powers. Weapon testing and fall-out have been the subject of considerable discussion in the world's press, with the expression

of widely different opinions by eminent scientists and statesmen. Such press 'campaigns' often culminate in petitions for the suspension of tests—petitions which are based on humanitarian considerations but which ignore the political situation. It is unfortunate that the public, whether scientist of layman, is not sufficiently well informed concerning the actual dangers of present levels of fall-out to really know whether such petitions are warranted.

Dr. Muller, a geneticist of Indiana University has estimated[1] 100,000-1,000,000 genetic injuries as a result of nuclear explosions to date. Dr. Pauling, a Nobel prize-winner, estimates[2] 1,000,000 deaths attributable to fall-out, and a Committee of the Atomic Scientists Association under the chairmanship of Professor Rotblat conclude[3] that each Megaton (explosive power) equivalent of fission products will be responsible for 1,000 cancer cases. This committee also warns of an indeterminate number of fatalities from leukaemia.

But are these prognostications a true reflection of fall-out potential for harm to man? This paper is an attempt to collate the available information and to obtain a balanced appreciation of the fall-out situation. To do this, it will not be necessary to extend the discussion far beyond the domain of radiation physics. I make this point because there are those[2] who are sceptical of the competency of physicists to pronounce on a matter which impinges on medicine, biology, genetics and other specialized disciplines. This attitude was expressed in a recent issue of an English medical journal[4].

'The Nuclear Boffins (God bless 'em all)
Have the 'fall-out' assessed to a decimal,
Yet my nephew and niece
Have got five legs apiece
And their intellect's infinitesimal'

It will become clear in the following discussion that any assessment of radiation hazards is based on the quantitative work of the 'nuclear boffins'.

Fission Products

Nuclear weapons are based on the energy release which accompanies the fission of uranium and other heavier elements. Fission is initiated by the absorption of a neutron and results in splitting the uranium (say) nucleus into two roughly equal parts—but there are a large number of possible primary pairs. These primary fission elements contain too many neutrons, and these are rapidly jettisoned with the formation of longer lived radio-active elements, which in turn decay, of half lives ranging from seconds to many years, with the emission of β or γ radiation. The secondary neutrons thrown out of the primary fission products are available to initiate fissions of other uranium nuclei. If the size of the uranium mass is large enough so that at least one of the secondary neutrons results in another uranium fission, the mass is said to be supercritical; a very rapid chain reaction will occur and the enormous energy release results in the explosion. About 10^{24} radio-active atoms are formed by a small A-bomb, vaporized with the material of the bomb and spread through the resultant fire ball.

If land or structural surfaces are enveloped in the fire ball, these too will be vaporized and carried up with the radio-active cloud. The fire ball is borne upward at a great velocity by its own buoyancy and any rubble in the neighbourhood will be carried up by the air currents in its wake. The fire ball cools as it rises and the vaporized matter, which includes the radio-active fission products, condenses onto or into condensed (or rubble-dust) particles of varying size from fractions to hundreds of microns in diameter. When the upward momentum of the cloud has been spent it spreads out laterally into the typical mushroom shape. Heavier particulate matter will begin immediately to drop back to earth under the influence of gravitational forces but lighter particles will drift with the existing air currents and might circle the earth several times before 'fall-out'.

TYPES OF FALL-OUT

We can distinguish 3 types of fall-out, viz. (*a*) local, (*b*) distant or tropospheric, and (*c*) delayed or stratospheric fall-out.

Local fall-out is intensified by the detonation of the bomb near the ground, when perhaps 80% of the fission products are deposited within a few hundred miles of the site of detonation. Local fall-out is virtually complete in a period of hours.

Distant or tropospheric fall-out consists of radio-active particles released into the troposphere, which are too small to be significantly affected by gravity. These particles will drift thousands of miles with the prevailing winds and circle the earth several times before deposition to the earth's surface is complete. The deposition process takes several months. It has long been known that deposition occurs primarily in rain.[6] First it was thought that the falling rain scoured the atmosphere on its way down, but another theory[7],[8] has recently gained favour. This suggests that the fall-out particles in random motion in rain clouds will eventually collide with and stick to the water droplets constituting the cloud. When it subsequently rains, the radio-activity is also precipitated. In this way, the atmosphere is periodically cleansed of particulate matter.

The fact that the tropospheric deposition is relatively rapid, combined with the existence of the belt of stable air over the tropics, confines this type of fall-out to the general latitude of the bomb test. Thus relatively little of the fission products released into the northern troposphere is deposited in southern latitudes.

Delayed or stratospheric fall-out, as the name implies, is that radio-activity, attached to small particles (some of which may be near molecular size), released into the stratosphere and deposited from it at a very slow rate. The troposphere or lower atmosphere extends to a height of 30-40 thousand feet and contains all the phenomena which we associate with our weather. The stratosphere or upper atmosphere is metereologically differentiated from the troposphere, and mixing between stratospheric and tropospheric air is very slow. Thus it has been found that radio-activity released into the stratosphere percolates through the tropopause at a rate of about 10%[9] of stratospheric storage per year. Sampling the atmosphere for fission-product radio-activity[6] has shown a precipitous increase in content per unit weight of air at 30-40 thousand feet—convincing evidence of stratospheric storage.

Not all nuclear weapons release fission products into the stratosphere. The power of the bomb must be sufficient to thrust the cooling radio-active cloud up into the stratosphere. An air-burst bomb of greater than one Megaton power equivalent is required.[5] Smaller bombs will give rise to tropospheric fall-out and local fall-out if ground burst.

Because most of the fission products decay quite rapidly, the harmful potential of the fission-product radio-activity is largely neutralized while still in the stratosphere and before it can do harm to the human race. For this reason, a high powered bomb burst at an altitude of 15 (or more) thousand feet is a relatively 'clean' bomb compared to the same power bomb detonated near the ground. But in contrast to tropospheric fall-out, stratosphere fall-out is expected to diffuse throughout the stratosphere and deposit rather uniformly all over the earth's surface.

ASPECTS OF FALL-OUT HAZARD

The radio-active fission products are finally deposited on the earth's surface, where they accumulate as a surface distribution of radio-activity additional to the naturally occurring distribution of radio-activity. This additional external irradiation to which man is exposed is much too small to result in observable harm such as effects on the basal layer of the skin or changes in the cells of the circulating blood. But it is believed that no amount of radiation dose is too small to cause genetic mutations. (A genetically effective dose is an average dose weighted over the whole population according to their probability of further reproduction.) Fall-out exposes the whole population and hence could conceivably be a *genetic hazard* even though the individual dose is small. It is thought, moreover, that man has probably reached an advanced state of natural balance such that all mutations can be regarded as potentially harmful. This might be a pessimistic view, but it clearly indicates the importance of a study of genetic damage resulting from an increased exposure of the human population —whether it be to fall-out or other man-made sources of radiation.

This ground deposition of radio-activity fall-out may also be absorbed into plants and animals and, through the human food chain, be ingested into the human body. If fixed into the body, the intimate contact of radio-activity and body tissue makes the radiation much more effective in premature aging of that tissue and it may induce the development of *cancerous diseases*. From this point of view, radiostrontium 90 is the only fission product which requires consideration. Strontium 90 is produced in about 5%[10] of fission reactions, has a very long half life of 28 years, behaves chemically like calcium and hence is absorbed with Ca into the skeleton, where a fraction will remain for many years.[11]

The β-radiations emitted by strontium 90 and its daughter product yttrium 90 are sufficiently penetrating to irradiate bone tissue *and* bone marrow (a blood forming organ) but are too rapidly absorbed to be able to affect the rest of the body. A bone concentration of strontium 90 is thus able to (*a*) increase the probability of the formation of *bone cancer* and (*b*) increase the probability of induction of a *blood 'cancer'* (leukaemia).

From the above paragraphs, it follows that the extent of the human damage caused by fall-out can be gauged from (1) the magnitude of the genetically effective external radiation dose from the *ground deposits* of radio-active fission products and (2) the dose to the bone and bone marrow from the *radiostrontium concentration in bone*.

Fall-out Measurement

In principle, the measurement of fall-out radio-activity is straightforward. One method in routine use in many parts of the world, including South Africa,[12] exposes a foot square of gummed film with a routine change every 24 hours. Fall-out particles carried by wind and rain have been found to stick to the gummed paper with quite high efficiency.[9] The measurement of the captured radio-activity is achieved by burning the organic material in a crucible in an oven at 500° C and transferring the ash residue to a holder placed under the thin-end window of a geiger counter. Extraneous contamination by naturally occurring radio-active elements must be avoided or corrected for and the counting efficiency of the geiger counter has to be ascertained by separate calibration.

Another collection method employs a high-sided flat-bottomed pot containing a layer of water to retain fall-out particles which enter the pot. Unlike the gummed film, the retention efficiency of the pot does not decrease with time and it is therefore better suited to the collection of long-period (say 1 month) accumulations of fall-out material. The 'pot-collections' are also better suited to

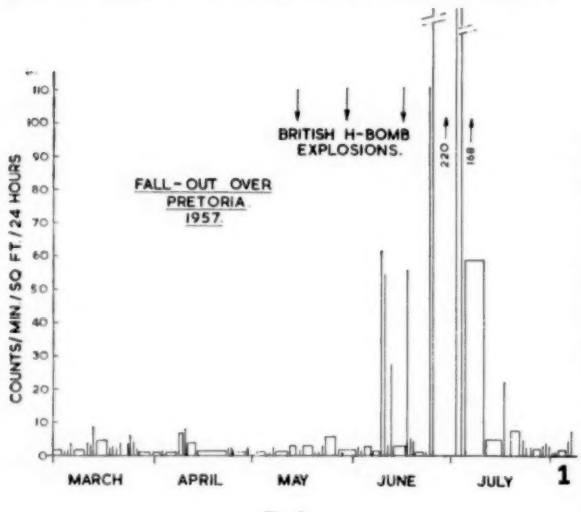

Fig. 1.

radiochemical analysis, particularly to determine the quantity of strontium 90 in the fall-out during the period of collection. Routine fall-out measurements have been made at the South African National Physical Research Laboratory since early in 1956. A sample of recent measurements of total activity is contained in Fig. 1.

Other important measurements of the total accumulated activity in the top soil, and the content of radiostrontium in the soil,[13] in food products and in human bone,[15] require careful radiochemical analysis to separate fission products from natural activities which would otherwise mask the fission-product activity.

I have attempted to summarize much of the available fall-out data in Table I. Such a condensation of information must necessarily be no more than approximate—but this will serve our purpose here. Local meteorological conditions vary considerably

TABLE I. FALL-OUT FACTS, 1957

Stratospheric Storage		30 m tons of Fission Products.
Deposition Rate		≡15 mc/mile² Sr^{90}, Cs^{137}.
		1·5 mc/mile²/year
(British Tests)	SOUTH	(American and Russian Tests) NORTH
Ground Deposition:		
50 mc/mile²		500 mc/mile² Total
8 mc/mile²		25 mc/mile² Sr^{90}, Cs^{137}

and, to a lesser extent, so also do fall-out figures, which are further affected by the latitudes most commonly used for nuclear weapon testing. The figures quoted in Table I are also extrapolations which attempt to include the most recent weapon explosions and they are probably higher than average for inhabited areas.

Another group of data is summarized in Table II. This concerns the human bone uptake of strontium 90.[5, 16, 17] Because of the similarity in behaviour of strontium and calcium, the concentration of strontium 90 is given as a function of calcium content ($\mu\mu$c Sr^{90} per g. Ca) and the uptake of strontium 90 is given relative to that of calcium. It is noted that in calcium-deficient soils, strontium

TABLE II. STRONTIUM 90 UPTAKE IN HUMAN BONE *

1 Strontium Unit (S.U.) = 1 $\mu\mu$c Sr90/g.Ca

1. *Food Chain*

Soil	Adverse Ca deficiency factor up to 5
Plant	1½–3 times Ca/Sr relative to soil
Milk	10 times Ca/Sr relative to soil

(Man derives 70–80% of calcium needs from dairy products)

2. *Bone Uptake*

Digestive System.	
Blood	Sr/Ca discrimination 2–3
Bone	Sr/Ca discrimination and preferential excretion of Sr

(Equilibrium discrimination of about 3)

3. *Over-all Discrimination Factor*

Sr/Ca in soil relative to Sr/Ca in bone

Normal Ca diet 60	(say 6–30)
Ca deficient diet 12	

uptake can be increased by as much as a factor 5. On the other hand, the biochemistry of plant and animal systems might discriminate against strontium by a factor 60. These discrimination factors are not known with any precision but additional information is being obtained at an accelerated pace.

About 600 bone samples were procured in 1955 and analyzed for strontium content.[16] The results have been detailed in Fig. 2. In 1955 the mean ground deposition was about 8 millicuries per square mile. Assuming that 50% of this was retained in the top inch of soil, which contains, on the average, about 75 g. of calcium per cubic foot, a strontium to calcium discrimination factor of 30 (see Table II) leads one to expect an equilibrium bone concentration of 0·8 S.U., which is not far above that observed in young children in 1955.

Continued Stratospheric Deposition

The existence of the stratospheric storage ensures a continued deposition of long-lived stratosphere fall-out (particularly Sr^{90} and Cs^{137}) for decades after all nuclear-weapon testing has been discontinued. At the present time there is about as much radiostrontium and radiocaesium in the stratosphere as there is on the ground and the recent emphasis on keeping bomb testing 'clean' has resulted in high altitude testing with consequent additions to the stratospheric store. This procedure in all future tests will certainly reduce the genetic hazard of fall-out (see below) while the

* *Note by author* (19 *November* 1957). I have recently read the detailed report of the 'Hearings before the special subcommittee on radiation of the Joint Committee on Atomic Energy Congress of the United States' (May-June 1957) on the 'Nature of radio-active fall-out and its effects on man'. In this two-volume report there is considerable contention on points of detail but the general impression is one of agreement on outline with uncertainty on the precise estimation of the future Sr^{90} hazard. The most recent results make some changes to the basic data for Sr^{90} hazard calculations, but the use of earlier information, as in my text, does not lead to faulty conclusions and a revision of the text does not seem to be warranted.

The discrimination factor of Sr^{90}/Ca from Soil to Bone is now variously placed between 10 and 20 and not 60 as in Table II (derived from the best of earlier estimates). In fact my text uses a value of 30 in all subsequent calculations so that the results are not more than a factor 3 on the optimistic side. This is to be weighed against a generally pessimistic choice of other factors.

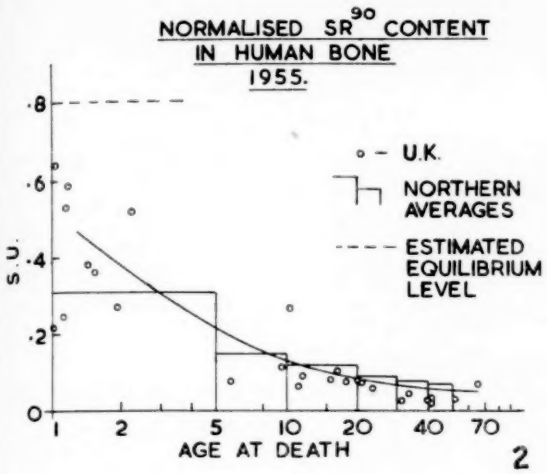

Fig. 2.

major effect on the strontium-ingestion hazard is to dilute the deposition by making it more uniform over the earth's crust.

Deposition Activities at Equilibrium

As a result of high-altitude testing and stratospheric storage the total surface deposition of fission-product radio-activity is likely to decrease rather than increase, even should further high-altitude testing occur. The activity of strontium 90 is, however, being continually replenished from the stratosphere, and if deposition (10% of the stratosphere store per year) is greater than the decay (2½% per year) of the strontium already deposited on the ground, the level of strontium activity will build up until this equilibrium is reached. If there are no further bomb tests this equilibrium will occur in about 10 years' time. If bomb tests are not discontinued, the equilibrium will be much further delayed.

The calculation (Table III) of equilibrium values is not a difficult one. I make the assumptions that (if weapon testing continues) the testing rate will be constant, most of the strontium goes initially into the stratosphere, and the concentration of strontium in bone is proportional to the ground deposition (see Table II).

TABLE III. FALL-OUT FUTURE

Future: Weapon Testing Rate	*Region*	*Sr90, Cs137 at Max. or Equil.*		
		Stratosphere	*Ground*	*Bone Content*
Nil	North South	15 mc/mi² 15 mc/mi²	25 mc/mi² 10 mc/mi²	2·5 S.U. 1·0
10 M tons per year (as for past 5 years)	North South	50	200	20

Radiocaesium. Caesium 137 is about as long lived as strontium 90 and is also produced in large quantities by fission reactions. Thus caesium and strontium, the two longest-lived components of fission products are also the only fission products which will deposit from the stratosphere in significant quantities. Caesium 137 is a β- and γ-emitter but it is not retained by the human body and hence does not constitute an ingestion hazard. However its penetrating γ-radiation can add to the external irradiation genetic hazard, to which radiostrontium, a pure β-emitter makes a negligible contribution.

PART II. DOSE AND HAZARD ASSESSMENT

The damage caused by concentrations of radio-active fall-out can only be estimated from the radiation doses which result from their presence. The most commonly used unit of dose is now the 'rad', which corresponds to the absorption of 100 ergs energy per g. of tissue. There are other units of dose but in what follows, the rad or röntgen (r) can be considered as interchangeable.

External Dose to the Reproductive Organs

This section will not pretend to give a precise dose calculation. It will give an outline of the relevant considerations and arrive at an approximate answer.

The radiation dose rate from radio-activity spread on an extended plane surface can be both calculated and measured. Thus the dose from 1 curie per mile² of γ-emitting radio-activity emitting 0·7 Mev photons is about 30 milli-r per year at a height of 1 metre above the plane—the mean energy of γ-radiation from mixed fission products is quite close to 0·7 Mev, as is also the energy of radiation emitted by caesium 137. We can therefore assume the above deposition-dose relation in the following calculation.

But not all the deposited fission-product activity will be fully effective. A proportion will be washed away or into the ground surface of inhabited areas, and persons are partially protected from this ground deposition by the buildings in which they live and work. A weathering factor of 2 or 3 (say 2·5) accounts for the former effect and a structural shielding factor between 2·5 and 7 (say 5) has been estimated for the latter.[18, 19]

It has already been stated that the effort toward 'cleaner' bombs and the decreased rate of testing will combine to cause a future decrease of total fission-product deposition. In the northern hemisphere there are high spots of 500 mc/mi² (Table I). If all future H-bombs of type similar to those already fired are detonated so that the major part of their activity goes into the stratosphere, a firing rate of 10 M tons per year will produce an equilibrium (max.) ground deposition of γ-emitting caesium 137 of 200 mc/mi² (Table III). In the southern hemisphere the present high-spot deposition is nearer 50 mc/mi² of mixed fission products but stratospheric fall-out from the above 10 M tons per year would increase this to at least 200 mc/mi² because of the caesium 137.

Taking the highest ground deposition which seems likely (i.e. the existing 500 mc/mi²), the genetic lifetime (30 years) dose from fall-out deposition is

$$(30 \times 0{\cdot}5 \times \tfrac{1}{2\cdot5} \times \tfrac{1}{5}) \times 30 = 35 \text{ milli-r/30 years.}$$

Genetic Hazard

Man has always been exposed to sources of radiation which add up to what we term the natural background. This includes cosmic rays, naturally occurring radio-elements of the Uranium, Thorium and Actinium series and more isolated long half-life isotopes such as potassium 40.

Radio-biologists will admit that our knowledge of the effects of this background radiation is insufficient to enable any assertion concerning its value (whether positive, negative or neutral) to the existence and evolution of man. It is known that all radiation, however low in intensity, is capable of causing genetic mutations and in the absence of more complete evidence it must be assumed that any mutations are likely to be undesirable. Thus it is the considered opinion of national commissions[18, 20] which were set up to study these questions that all radiation must be regarded as genetically undesirable and they emphasize that genetic effects *are* proportional to the total radiation dose.

However, ionizing radiation is not the only cause of genetic mutations and the natural mutation rate is augmented by the effects of heat and chemicals. It has been suggested[20] that this burden of mutations is a sort of normal burden on society which we may eventually be able to partially control. Human genetics is a very complex subject and its study is complicated by the meagre data available for man. Extrapolations from plant, insect and mammal studies have been made by experienced geneticists, who independently in the U.S.A. and the U.K. concluded that the mutation-rate doubling dose is between 30 and 80 r—or about 7-20 times the average genetic lifetime radiation dose from natural sources. In other words, radiation background is responsible for 1/7th to 1/20th of the natural mutation rate. The evidence is so inadequate that even these limits are not known with certainty.

Our only index of genetic damage is the totality of tangible genetic defects such as mental defects, malformations, epilepsy, neuromuscular defects, defects of vision and hearing, etc. Roughly 4-5% of all live births have defects of this kind, and of these perhaps half (2%) have a simple genetic origin.[20] It is relevant to note here that experiments on animal and vegetable populations show that there is a constant elimination of unfavourable factors by inhibition of fertility and that in one investigation[21] alteration of the structure of the chromosomes (genetic damage) produced inhibition of reproduction in 98% of the cases examined.

The information presented above has been used in Table IV to calculate the genetic effects of fall-out. This takes us back to those who issue dire warnings of the ill effects of nuclear weapon testing. Their calculations are obviously genuine but are they not guilty of a lack of a sense of proportion? It is immediately noticeable

TABLE IV. CALCULATION OF GENETIC DAMAGE ASCRIBABLE TO FALL-OUT

1. Natural Background Dose to age 30 (4r)
2. Genetically effective fall-out dose, 0·04r (0·1r)
3. Natural Radiation responsible for 1/7 to 1/20 (say 1/10) existing genetic damage
   Hence fall-out responsible for $1/10 \times \cdot 04/4 = 1/1{,}000$ existing damage
4. 2% of live births have simple genetic defects
5. Increased genetic load for population $1/1000 \times 2/100 \times 3 \times 10^6$
   60,000 defective persons

that the fall-out dose is about 1% (some estimate 2 or 3%) of the natural background. This background dose is not precisely the same everywhere, and when closer study is given to the existing variations in natural background radiation doses[22, 23] the facts presented in Table V emerge.

TABLE V. GENETIC DOSE FROM NATURAL SOURCES

Compare: Fall-out dose rate ·003r/year

1. Altitude effect, cosmic rays
   Dose rate increase of ·003r/year/1000 feet
   At high altitudes ·03r/year/1000 feet
2. Architectural effect
   Out of doors ·05—0·1r/year
   Wooden house 0·06r/year
   Concrete, brick 0·08—0·1r/year
   Concrete, granite 0·1—0·17r/year (Max. over 0·5r/year)
3. Diagnostic X-radiology
   Population gonad dose ·02—0·15r/year
4. Luminous watch (0·2μc Ra per watch)
   Gonad dose 0·01r/year.

The obvious conclusions from the information given in Table V are that (*a*) the fall-out contribution to the dose to the gonads is negligible and not likely to become otherwise (always assuming the avoidance of a large-scale nuclear-weapon war) and (*b*) if the deleterious genetic effects of small doses of radiation are a matter for concern, we should find an excuse for living permanently at the seaside in wooden houses. This would, of course, be both impracticable and foolish. But we can take all reasonable care in the use of X-rays, radium and radio-isotopes. There is certainly no cause for limiting the use of radiations which have been and will be of very great benefit as a tool in medicine, industry, agriculture and research. At the same time, it will be necessary somehow to compromise between minimizing our exposure to radiation while maximizing the benefits it offers to our health and material well-being.[24]

The Bone and Blood 'Cancer' Hazard

The radiations emitted from deposits of strontium 90 in the bone can cause bone cancers while the irradiation of the bone-marrow might result in laukaemia ('blood cancer'). Animals injected with strontium 90 have developed bone and blood 'cancers' and in humans, radium has been observed to increase the incidence of bone tumours. In all these cases the amount of radio-activity present was much larger than is likely to result from ingested fall-out. Here lies the fundamental obstacle to a certain appraisel of the effects of the fall-out; the relationship between dose and effect—for small doses—is not known with certainty. It may be that radiation-induced cancers are a threshold-type reaction for which small quantities of ingested radio-activity would not result in harm. On the other hand, the effect may be proportional to dose (like genetic mutations) and then even the smallest additional irradiation will increase the incidence of these diseases.

Writing of *tumour induction*, the Medical Research Council conclude that 'on the whole the experiments seem in favour of a proportionality between the frequency of tumours produced in a given time, and the amount of radio-active material in the body even at low dose levels.'[16] The data from Hasterlik's survey[25] of lesions in bones of humans who have ingested varying known amounts of radium supports this proportionality conclusion and by extrapolation enables one to estimate the probability of tumour induction by a given amount of radium in the body. This forms the basis for the calculation of bone cancer (Table VI) due to ingested strontium 90. The method of calculation is that used by Professor Rotblat and a committee of the Atomic Scientists

TABLE VI. CALCULATION OF BONE TUMOURS FROM STRONTIUM INGESTION

1. From animal experiments: relative toxicity of ingested radium and strontium such that
   $0{\cdot}1\mu c\ Ra \equiv 1000\ S.U.\ (1\mu c)\ Sr^{90}Y^{90}$
2. 0·1 μc ingested Ra gives a 0·5% probability of tumour production
3. Hence 10 S.U. of Sr^{90} results in
   $10 \cdot 1000 \times 0{\cdot}5 = \cdot 005\%$ probability
   i.e. 150,000 cancers in world population

Association.[3] Hasterlik's data show that the incidence of so-called major lesions is 10 times as great as the incidence of malignancies (cancers). This does not necessarily mean that the damaging effects of strontium 90 are grossly under-estimated by the calculation in Table VI. It is a known fact that tumours are more readily induced by localized irradiation. As the energetic alpha particles emitted by radium (and its daughters) have a range which is less than 1% of that of the β emission from strontium (and yttrium, its daughter), it is quite probable that the tumour-producing capabilities of strontium are being over-estimated.

What of Leukaemia?

The data on the occurrence in man of leukaemia caused by ionizing radiation also favours a simple proportional relationship.[26, 27] Evidence[28] also indicates that doses as small as those received by the foetus from radiological examination of the pregnant mother's abdomen are leukaemogenic. Such doses are in the range of a few röntgens per examination.[29]

The assumption of a simple proportional relation between dose and the incidence of leukaemia implies again that any radiation dose, however small, will be effective. This might be relevant to the fact that in many countries the annual death rate from leukaemia is rising.[26] Some of this rise is probably genuine and may, in part, be due to the very rapidly expanding use of ionizing radiations for diagnostic purposes. Osborn and Smith[29] have estimated that 17-18 million X-ray examinations are performed annually in England and Wales and this rate is increasing by 12% each year. I am not here advocating a reduction in the medical use of ionizing radiations. I am stating the case for a more controlled use of this invaluable tool. It must always be up to the doctor to decide whether the harmful potential of ionizing radiations outweighs the results which the use of these radiations may achieve for the particular patient. The doctor must, of course, be well informed about the effects of radiations.

If even small amounts of general irradiation can be leukaemogenic, it must follow that those who are occupationally exposed to radiations are exposing themselves to additional risk. Here again, a sense of proportion is vital to a proper appreciation of the harmful potential of artificial sources of radiation. It is undoubtedly true that everyday life holds many hazards such as the motor car, tobacco smoking, nervous strain, and even self-indulgence in food and drink, which far exceed the hazards encountered in the radiation laboratory—let alone those incurred as a result of exposure to radio-activity in our environment.

The presently available information on radiation-induced leukaemia has been collated and discussed by Professor Lewis.[27] The evidence from five different sources (not all quantitatively reliable) is surprisingly consistent and strongly implies the proportional relationship between dose and effect. Lewis derives a probably of leukaemia of specified type per individual per unit of absorbed dose in the bone marrow. The best estimates of this quantity lie in the range $1\text{-}2 \times 10^6$ per rad which corresponds to an incidence-doubling dose between 25 and 50r (the present incidence is near 50 per million population). As the dose from natural background is about 4r in the average life expectancy (about 30 years) it follows that natural background is probably responsible for about 1/10th of the existing incidence of leukaemia fatalities. It is interesting that the contribution of natural background to existing genetic mutation effects is also close to 1/10th. One is consequently tempted to think that the mechanism of leukaemia induction could be a simple somatic mutation. In fact the mechanism cannot be so simple[30] because protection of some of the bone-marrow from irradiation very considerably reduces the induction of leukaemia.

The calculation of bone-marrow dose from bone fixation of radiostrontium is given in Table VII. It is based on the assumption that the strontium is uniformly deposited in the surrounding bone. The calculation in Table VII does not account for screening of the bone marrow from the short-range β-emission. This screening

TABLE VII. CALCULATION OF Sr^{90}—Y^{90} BONE-MARROW DOSE

1. 1 S.U.$\equiv 10^{-9}$ μc Sr^{90} in 10^4 g. bone (average human)
2. Energy emitted per Sr^{90} disintegration
   $= (0{\cdot}54 + 2{\cdot}24)$ Mev $= 4{\cdot}5 \times 10^{-6}$ ergs.
3. Absorbed dose unit
   1 rad = 100 ergs/g.
4. Absorbed dose from Sr^{90} ingestion
   $$= \frac{\text{(dis./year) (energy/dis.)}}{\text{(weight of bone)} \times 100} \text{ r/year}$$
   $= 5$ mr/yr/S.U.
5. Bone surrounds marrow. Perhaps only 50% irradiation of marrow, i.e. 2·5 mr/yr/S.U.

would be increased by a non-uniform distribution of the strontium in the bone, but decreased if concentration of activity occurred near bone-marrow regions more actively concerned with blood-cell production.[16] On the other hand there is the already mentioned correlation between leukaemia incidence and the volume of irradiated marrow tissue;[26, 30] so that increased dose near localized

TABLE VIII. CALCULATION OF LEUKAEMIA FATALITIES

1. Max. expected Sr^{90} bone conc., −10 S.U.
2. Marrow dose = 0·75r in 30 yr. life expectancy.
   Compare:
   Marrow dose from average background
   = 3·6r in life expectancy
   = dose from 50 S.U. of Sr^{90}
3. Probability of radiation-induced leukaemia
   2×10^{-5}/r
4. Leukaemia from ingested Sr^{90}
   $0{\cdot}75 \times 2 \times 10^{-4} \times 3 \times 10^{9}$
   = 4,500 deaths per generation.

strontium concentrations is probably more than balanced by the decreased dose to the rest of the marrow. This means that the assumption of uniformly distributed strontium is likely to lead to overestimation of the effects of ingested strontium on the incidence of leukaemia (Table VIII).

The calculation of Table VIII uses a bone concentration of 10 strontium units. This value of 10 S.U. was taken as a suitable round figure near the likely *maximum* for the case of no further bomb testing and near the equilibrium *average* if testing of weapons continues at the rate of 10 Megatons per year (see below).

It is noteworthy that 10 S.U. gives a dose to the bone which is about 1/5th that from natural sources of radiation. The dose from the average content of radium in the bone corresponds to that from about 15 S.U. The radium content of bone also depends on environment and may be twice the average value. Furthermore, as has already been stressed, the external sources of natural background radiation vary in intensity in such a way that a variation of background dose by a factor 2 or 3 either way is not unusual and, in extreme localities, the background may increase by a factor up to and greater than 30. I repeat these facts in order to make it clear that a simple comparison of fall-out doses with the dose from an average background over-emphasizes the relative importance of the fall-out hazards.

The facts already presented enable one to make a fairly detailed over-all assessment of the strontium hazard, present and future. It is not claimed that no uncertainties remain. The most important of these lie with *individual variation* and the strontium/calcium *discrimination factor*. The highest concentrations will be found in isolated individuals who obtain their total food supply from a restricted area that has very low available calcium in the soil.[10] The effects of the *strontium distribution in the bone* seem inadequately understood and some uncertainty also remains concerning the *effect of age* on the strontium bone content. It is at present assumed that strontium content is independent of age for those who have lived all their lives in the same fall-out field. Keeping in mind the fact that the average background dose corresponds to about 50 S.U. of strontium in the adult skeleton, the situation concerning strontium ingestion from fall-out can be summarized as follows:

1. *Situation in* 1956.

Measured bone content. Maximum (stillborn) about 1·0 S.U. Average (adults) about 0·12 S.U.

N.B.—Bone dose about 1/400th of the average background dose.

2. *Situation in about* 1970, *assuming no further tests.*

In the North (e.g. U.S.A.) about 2·5 S.U.

In the South (e.g. South Africa) about 1·0 S.U. (Multiply by 5, perhaps, for calcium-deficiant diet.)

N.B.—Bone dose about 2-5% of an average background dose.

3. *Situation in perhaps* 40-50 *years, assuming* 10 *Mtons/year*

World wide about 20 S.U. (Multiply upward for Ca deficient diet.)

N.B.—Bone dose about 40% of an average background dose.

CONCLUSION

It has been shown that it is not a very difficult matter to make estimates of the hazards to man of radio-active fall-out from nuclear-weapon testing. Uncertainties remain but the general picture is clear. It turns out that the genetic effects of radio-active fall-out can be neglected. It is inconceivable that the fall-out radiation dose to the reproductive organs should ever be other than negligible relative to the background radiation dose. This conclusion would only be invalidated in the event of a nuclear-weapon war.

While the effects of bone concentrations of radiostrontium are still very small relative to the effects of background radiation, this aspect of fall-out hazards requires to be closely watched. The bone concentration is expected to continue to increase during the next 10 years, whether or not weapon testing is discontinued, but even 10 M tons per year of nuclear weapon explosions is not expected to raise the dose from fall-out strontium in the bone to that from an average natural background. No more need be said about the variations of this natural background dose.

Everyone is aware that radio-active fall-out has harmful potential. Many feel that in such circumstances, any nuclear-weapon testing is morally indefensible. In my view, however, the question of weapon testing cannot be divorced from the political situation and in this connection the words of the late Professor Lord Cherwell are very relevant. In his typically outspoken manner he pronounced[2] on 'hysterical people' who would sacrifice 'a deterrent which would probably save us from a war costing millions of lives' on the grounds that 'our tests might harm the health of a completely negligible part of the human race'.

SUMMARY

The facts concerning radio-active fall-out from the testing of fission-type nuclear weapons are reviewed and summarized in a form convenient to the assessment of the resultant effects on man. These are seen to be twofold, viz. (1) the genetic effect and (2) the effects of ingested strontium 90.

The genetically effective dose from fall-out is compared with the very variable naturally occurring background radiation dose and it is concluded that the genetic aspect of fall-out hazard is relatively unimportant. The remaining uncertainties in the estimates of the effects of ingested strontium 90 are not sufficient to completely invalidate conclusions based on the available data. It is shown that should nuclear-weapon testing continue at its present rate of 10 Megatons per year, the dose of strontium 90 to the bone should not exceed the average dose from natural sources. The strong dependence of the intensity of natural background on the human environment is emphasized as a very relevant consideration.

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REFERENCES

1. Report from Chicago, *Pretoria News*, 5 June 1957.
2. *How dangerous are the bomb tests? Time*, 3 June 1957.
3. Report of the Atomic Scientists Association (1957): Lancet, **1**, 878.
4. Verse (1957): *Ibid.*, **1**, 882.
5. Libby, W. F. (1956): Proc. Nat. Acad. Sci. (Wash.), **42**, 945.
6. Stewart, N. G., Crooks, R. N. and Fisher, E. M. R. (1955): Rep. Atomic Energy Res. Est. HP/R 1701.
7. Libby, W. F. Address to the American Meteorological Society, Chicago, March 1957. US AEC.
8. Greenfield, S. M. (1956): Rep. Associated Equipment Co. US AEC rep., p. 883 (July).
9. Libby, W. F. (1956): Proc. Nat. Acad. Sci. (Wash.), **42**, 365.
10. Plutonium project (1946): Rev. Mod. Phys., **18**, 513.
11. Effective half life of 2,700 days. Recommendations of the International Commission on Radiological Protection (1955): Brit. J. Radiol. Suppl. 6.
12. S. Afr. Coun. Sci. Indust. Res. (1957): Res. Rev. **7**, no 1 (March), p. 10.
13. Harley, J. H. (1956): U.S. Hlth Sfty Lab. Rep. 56-10, 15 Oct.
14. Bryant, R. J., Chamberlain, A. C., Morgan, A. and Spicer, G. S. (1956): Rep. Atomic Energy Res. Est. HP/R 2056.
15. Martell, E. A. (1956): Rep. Associated Equipment Co. US AEC rep. U3262 (May).

16. Kulp, J. L., Eckelmann, W. R. and Schulert, A. R. (1957): Science, 125, 219.
17. Harrison, G. E., Raymond, W. H. A. and Trethaway, H. C. (1955): Clin. Sci., 14, 681.
18. *The Hazards to man of nuclear and allied radiations* (1956): Spec. Rep. Ser. Med. Res Coun. (Lond.).
19. Rotblat, J. (1956): *Biological Hazards of Radiations. Times* (Lond.). Science Review, Autumn (1956).
20. *The biological effects of atomic radiations* (1956): Nat. Acad. Sci. (Wash.).
21. Bilquez, A. (1957): Conseil scientifique pour l'Afrique. Specialist meeting on Radio-isotopes held in Pretoria, July.
22. Spiers, F. W. (1956): Brit. J. Radiol., 29, 410.
23. Libby, F. W. (1955): Science, 122, 57.
24. Tayler, L. S. (1957): Amer. J. Roentgenol., 77, 914.
25. Hasterlik, R. J. (1956): Geneva Conf. Peaceful Uses Atomic Energy, vol 2, p. 149. New York. UN.
26. Court-Brown, W. M. and Doll, R. (1957): Spec. Rep. Ser. Med. Res. Coun. No. 295.
27. Lewis, E. B. (1957): Science, 125, 965.
28. Stewart, A., Webb, J., Giles, D. and Hewitt, D. (1956): Lancet, 2, 447.
29. Osborn, S. B. and Smith, E. E. (1956): *Ibid.*, 1, 949.
30. Alexander, P. (1957): *Atomic Radiation and Life*, p. 153. London: Pelican Books.

THE VIEWPOINT OF A MENTAL HOSPITAL PSYCHIATRIST ON THE PROBLEM OF ALCOHOLISM*

A. McE. LAMONT, M.D. (GLAS.)

Assistant Physician Superintendent, Weskoppies Hospital, Pretoria

Chronic alcoholism is almost invariably rooted in mental abnormality and should thus be regarded as a mental disease. The problem cannot be separated from public-health endeavour in the field of mental hygiene. Those of us working in psychological medicine are very conscious of the isolation of our speciality and we look forward to closer collaboration and help from our colleagues in public health. We feel confident that the machinery operating so efficiently in the prevention of physical diseases could play a similar role in combating mental illnesses. Alcoholism is an obvious condition with which to commence measures in preventive psychiatry.

Bowlby[1] has assembled a formidable amount of evidence to support the view that the child's experience in his family in his early years is of central importance for his healthy emotional development. Trained professional personnel should direct their energies to the prevention of 'family failure', which undoubtedly is an important contributing factor to the development of the pathological use of alcohol in susceptible individuals. These people find that alcohol can quiet their feelings of insecurity and inner conflicts, for the time being at least. An extension of child-welfare services by the establishment of *child guidance clinics*, run by child psychiatrists would make a contribution to the prevention of alcoholism. Trained health visitors and social workers should be aware of the emotional needs and motivations of individual families that come under their care. The local medical officer of health is the man who should plan and control these endeavours. The first step is to start with training in the principles and practice of psychological medicine for all public-health personnel. A start has been made in Johannesburg with a course for health visitors and, no doubt, other centres will follow. The organization of the attack on alcoholism should be based on the discipline of psychiatry. It should not be left to well-meaning amateurs. Advantage should be taken of the wealth of clinical material in our mental hospitals for the training of professional staff.

Alcoholism can be symptomatic of other psychiatric entities. Thus all alcoholic cases should be assessed in the light of the whole of clinical psychiatry. The ideal is for each case to be seen by an expert in psychological medicine, but tremendous headway will have been made when all general practitioners have had the training to recognize minor and major mental maladies in their early stages. I have acted as visiting psychiatrist to Sonderwater work colony for some years and have seen many cases of schizophrenia, manic depressive psychosis, border-line mental defect and epilepsy among 'alcoholics'. Shortage of accommodation has made their transfer to mental hospitals and institutions for defectives a tedious process. All these cases had been seen by practitioners before their committal and it is unfortunate that they have to go to a work colony before their basic mental condition is diagnosed. If we were to use the medical staff of mental hospitals at out-patient departments in various parts of the country, perhaps we could make an impression on this problem. That is what they do in other countries. In the UK the trend is for senior mental hospital psychiatrists to spend more than half of their time seeing patients *before* they reach institutions.

The tendency is for alcoholics to be referred for psychiatric treatment only when they are in the late stages of chronic alcoholism, unless they develop a frank psychosis. Chronic alcoholism has a poor prognosis and we are failing in our duty as medical men if we leave the early alcoholic to become chronic before we treat him. Chronic alcoholism is attended by progressive intellectual and physical deterioration; its effect on family life and the emotional development of children can be devastating. The latter effect is self-perpetuating because the children will, more than likely, exhibit emotional difficulties, including alcoholism, when they grow up. Again its role in absenteeism, accidents, suicide, homicide and sex crimes is well known.

The practical importance of the psychiatrically trained nurse in the management of the acute and chronic hospitalized alcoholic cannot be too strongly stressed. Anyone who compares psychiatrically disturbed patients in general wards and in psychiatric wards will be struck by the rapidity with which they settle down in the psychiatric wards. It is the psychiatrically trained nurse who does the trick. If these cases are to be treated in general hospitals then the ideal is to have the wards staffed by mental hospital nurses. The serious shortage of candidates qualifying as trained psychiatric nurses is one of our major public-health problems in South Africa today.

Under section 52 of the Mental Disorders Act, alcoholics can apply for admission to a mental hospital for a period not less than 6 months. Once the statutory form (M.S. 59) has been completed the period of treatment cannot be shortened except by the Commissioner of Mental Hygiene or the visiting hospital board. Results in cases treated under this section have been disappointing from a psychiatric angle although, of course, the physical condition of the patient improves greatly. The chronic alcoholic shows certain constant psychopathological features—he is emotionally immature and he shows a peculiar perversity of attitude towards authority. This is the reason why he builds up resentment against the rigid rules relating to his period of detention and almost invariably again resorts to alcohol when released.

Psychotherapeutic measures in chronic alcoholism require skilful management by a team in which psychiatrists, medical practitioners, psychologists and social workers must play a part. Short-term hospitalization with out-patient follow-up assisted by Alcoholics Anonymous is the best approach.

Alcoholics Anonymous has so far had the best results in re-socializing chronic alcoholics. However, it is not suitable for all cases and especially early cases who are loath to make the necessary initial admission of their inability to control their alcoholic habits. A comparison of alcoholism with tuberculosis may be useful. While it is important to care for advanced cases of tuberculosis in the community this is only a small part of a total preventive programme. From a public-health point of view the obvious aim is to prevent alcoholism in the community and treat the early alcoholic before he becomes chronic.

In the report of the Commissioner for Mental Hygiene we find that alcoholism is not a common diagnostic label among hospitalized psychiatric invalids. On 31 December 1955 there were 237 patients with a diagnosis of alcoholism out of a total of 18,919 in all institutions in the Union. Of these only 29 were being treated under section 52; the others were certified cases of alcoholic psychosis. Expressed as percentages, 1·25% of the total

* A paper read at a conference of the Union Health Department with full-time medical officers of health called by the Secretary for Health, Pretoria, 14-16 October 1957. Published by permission of the Secretary for Health.

were alcoholics. The male European had the highest percentage (3·81%) followed in descending order by female European (1·88%) male Coloured (0·89%), female Coloured (0·74%), male Native (0·48%), female Native (0·37%) and male and female Asiatic (0·00%). The incidence in new admissions is higher, however, It has been previously reported[2] that in 11·6% of male Bantu admissions to Weskoppies Hospital in 1952 alcohol played a major role in the aetiology of the illness.

REFERENCES

1. Bowlby, J. (1952): *Maternal care and Mental Health:* Wld. Hlth. Org. Monogr. Ser., no 2.
2. Lamont, A. M. and Blignault, W. J. (1953): S. Afr. Med. J., 27, 637.

A SOUTH AFRICAN DOCTOR'S IMPRESSIONS OF PUBLIC HEALTH IN THE UNITED KINGDOM*

DUNCAN L. FERGUSON, M.B., CH.B., D.P.H.

Medical Officer of Health, Port Elizabeth

Before leaving for overseas I was given a letter of introduction by Dr. le Roux, Secretary for Health, to the Ministry of Health in London. This enabled me to see a great deal in a relatively short time with the minimum of trouble, and I was afforded every possible facility and courtesy.

It was immediately obvious on my arrival in the UK that significant changes have taken place since 1919, when I commenced my medical studies in that country. The masses of the people are better housed and fed but, on the other hand, there is now much less evidence of luxury among the few than in my student days. As an instance of this, many large family dwellings have been partitioned and now accommodate two or more families instead of one.

Poliomyelitis

As my department had already, towards the end of 1955, given the first injection of South African polio vaccine to a number of White children in Port Elizabeth, it was interesting to see the procedure adopted in London early in 1956.

The vaccine used was placed on the market by a well-known firm of manufacturing chemists. Clear and concise printed notes for medical officers administering the vaccine were issued at a South London clinic, for instance. These ensured uniformity of technique and probably eliminated the inoculation of a certain number of individuals who may have been incubating the disease at the time of injection. It was important at that stage to avoid if possible putting any doubts in the public mind about the efficacy and safety of the vaccine. Parents were told that time was required to establish any degree of immunity. As in South Africa, public response was gratifying.

Tuberculosis and Diseases of the Chest

In a recent report of the Chief Medical Officer of the Ministry of Health, it is stated that 'within the last few years the scope and safety of thoracic surgery have been greatly widened as technical skill has developed, until at present the demand for thoracic surgical facilities has become one of the major problems the National Health Service has to meet'. This seemed to me to be an important statement for us in South Africa to bear in mind.

One of the mass radiography units which I visited was operated by the North-West Metropolitan Hospital Board and not by the local authority. 1,330 deaths due to tuberculosis which had not been notified before death were recorded in England and Wales in 1954. It is considered that mass X-ray surveys may to some extent eliminate this danger of unknown infective cases. Seeing that this problem is still of significance in England, it illustrates how much greater it is with us, who have to deal with millions of primitive people.

Freeze-dried (lyophilized) BCG vaccine for intracutaneous injection from the Pasteur Institute in Paris was being used in certain clinics on selected groups, e.g. contacts of notified cases, at a London clinic which I frequently attended. Team work is the keynote of anti-tuberculosis organization. The physician in charge of this particular clinic also had beds under his care in a general hospital in the area. This system ensured continuity of treatment.

Carcinoma of the Lung

Carcinoma of the lungs appears to be definitely on the increase; the increase is not merely apparent and due to improved methods of diagnosis and certification or a changing age-grouping of the population. It is commoner at present in urban than in rural areas and commoner in men than in women. Excessive cigarette smoking and pollution of the air by smog in crowded parts of bigger cities, are possible causal factors under investigation. References to the disease appear in the lay press as well as in the professional journals more frequently than in South Africa, and the disease is often mentioned by people in general conversation in view of the growing incidence among friends and relatives. In health education we should warn against excessive smoking and in town planning we should guard against possible air pollution by installing suitable machinery and so forth and by the proper siting of factories.

Venereal Diseases

Venereal diseases in the UK are commonest amongst criminals, prostitutes and newcomers to the country.

The clinics are relatively small and the treatment available very effective. Patients contracting the infection are examined in all respects on admission and again on completion of treatment. In one big London clinic patients presenting themselves after normal hours are attended to by the house surgeon or house physician on general duty, and privacy is assured.

Of 256 known prostitutes examined recently in a London prison, 36 had syphilis, 102 gonorrhoea and 76 other genital discharges. Non-specific urethritis is a very common complaint among males.

Fluoridation of Water Supplies

Dental services are well organized in the UK, but patients complain of the long waiting lists and the delay in obtaining appointments.

Consideration is being given to the problem of the elimination of dental caries and particularly to the advisability of the addition of fluoride to public water supplies in areas where fluoride does not occur naturally. The data available, particularly from America, indicate that in a concentration of approximately 1 part per million a reduction of 60% over a period of years in the incidence of dental caries can be expected. Excessive amounts of fluoride lead to unsightly mottling of the teeth and it is essential to avoid this. Watford, near London, which to all intents and purposes has no fluoride occurring naturally in its water has adopted the procedure of adding 1 part per million artificially and it was most instructive to witness this being carried out in actual practice. The apparatus used is relatively simple and does not require elaborate alterations to existing water filtration plants for its installation and it is comparatively inexpensive. Chemicals such as lime, alum, chlorine are as a routine measure added to public water supplies and there is really no problem in doing the same with fluoride.

Records are being kept of the D.M.F. rate or index, i.e. the incidence of decayed, missing and filled teeth, so that in years to come results may be assessed. The present position has been stated by Jean R. Forest, L.D.S., of the British Ministry of Health in the following terms: 'It has been established quite conclusively that naturally occurring fluoride ingested from drinking water effects a marked reduction in the incidence of dental caries and that with a concentration of about 1 part per million of fluoride in the water this reduction is of the order of 60%.'

There was, as expected, a certain amount of public disapproval in Watford of the proposal to add fluoride to the water supply and the whole matter was discussed freely through the press. The arguments against fluoridation seemed to be similar to those

* A paper submitted at the South African Medical Congress, Durban, September 1957.

used when pasteurisation of milk, immunization against various infections and so on, were introduced.

Hospital for Retarded Children

A visit to a 700-bed hospital for retarded children at Tooting was an eye-opener. Suitable school classes are provided in the hospital premises for those children who are educable to some extent. A neuropathological laboratory has been established for research purposes. The importance of infection with German measles of women in the early months of pregnancy was forcibly brought home to me on this occasion.

Maternity and Mothercraft Centres

The maternity and mothercraft centres were very interesting. Education plays a great part in their work. The following programme for every Thursday morning was published by one of these centres at Haverstock Hill, Hampstead:

'*Mothercraft Talks at Maternity and Child Welfare Centre.* (1) 'Mother to be' looks after herself. (2) Layette. (3) Bathing baby. (4) Breast feeding. (5) Milestones in a baby's life. (6) Weaning. (7) The bottle baby. (8) The nursing mother looks after herself. Demonstrations of gas and air analgesia will be given; also a film show'.

These clinics are run by local authorities. Marmite, orange juice, dried milk and many other articles were provided to mothers at greatly reduced rates. It was at these child welfare clinics and at the day schools particularly that one was forced to the conclusion that large groups of the recipients of these privileges did not fully appreciate their value and were not conscious of the almost intolerable burden of taxation the nation as a whole was called upon to bear to provide them. In our developing social services in South Africa, as distinct from purely health ones, it is well to bear these human weaknesses and tendencies in mind.

Health Education

Health Education in general is regarded in public health circles as one of the utmost importance. Some health departments in England have appointed special health education officers, the medical officer of health continuing to act as the guiding hand. In such matters, the health faddist who disregards the normal usages of the life about him, and the physical-exercise enthusiasts who spend so much time keeping fit that they have little time for anything else, must be subject to tactful restraint; a sense of balance must be maintained or else the public will be disappointed because the results will not ultimately be forthcoming. Health education must be in the hands of informed persons and must be regarded as an aspect of education as a whole; it is a life-long process.

General Sanitation

In at least one local authority area the crews of the refuse removal vans are paid a commission on the value of salvaged materials collected by them in the course of their duties. This, I was informed, was profitable both to the Borough Council and to the men themselves. In food handling, the responsibilities of the owners of businesses and factories, of employees and also of customers are emphasized. In our country the manner in which many customers handle empty milk bottles and articles of food in shops exposed for sale, leaves much to be desired.

Foot Survey

Care of the feet is a health activity of interest and importance in some areas. In Salford a foot survey was carried out in 1951 on 1,338 children, and gross defects were recorded as follows: Long arch weakness 3·7%, hallux valgus 2·3%, defects of lesser toes 3·7%. In addition, there were many cases with slight defects such as long arch weakness 13·8%, hallux valgus 4·6%, defects of lesser toes 6·9%.

It was concluded that ill-fitting shoes were responsible for many foot defects. Cooperation of parents with shoe retailers is essential. Chiropodists are employed by some public authorities. Foot hygiene is very important with elderly persons.

Crematorium

Sir Henry Thompson, F.R.C.S., pioneered cremation in England and there is a statue to him in the Golder's Green crematorium, London. Approximately 26% of the dead in England as a whole are disposed of by cremation. On the day I visited the Golder's Green Crematorium there were 17 cremations. The organization is highly efficient.

General

At Finsbury health centre there is an organization for employing aged persons for 2 hours per day, as well as providing other amenities for them. They are paid a nominal cash wage for work done. Absenteeism is very low. The persons attending strike up friendships and visit one another outside. On the day of my visit one group was engaged in filling small cardboard containers with wool on behalf of a wholesale chemist, who paid for the service rendered. Such work is said to help the mental outlook and attitudes of the old folk and to make them happier and more reasonable, than if they were entirely unoccupied.

Huge blocks of flats as well as cottage estates, have been erected by public authorities all over the United Kingdom to deal with the housing problem. Those visited were let at moderate rentals and provided all modern facilities to the occupants. They represent a great improvement on the old slum dwellings which they have mostly replaced and they have been so designed that there is a certain amount of open space around for playgrounds for the children.

The reconstruction of the centre of Coventry, heavily bombed during World War II, is already well advanced. Behind the shops are three-level car parks for 1,800 cars. The shops are around pleasant paved courts. The planning is such that the shopper can walk to her shop and do some window gazing sheltered by an overhanging upper storey. I suggest that shelter for the public in our bigger cities should receive more consideration than hitherto when master plans for shopping centres are being prepared.

The garden town of Bournville and the model factory of Cadbury Bros., were most impressive, particularly in regard to amenities provided for the workers, which include a beautifully constructed theatre, swimming baths and cafeterias.

In 1841, the expectation of life in England and Wales was for males 40 years and for females 42. In 1954, the figures were for males 68 years and for females 73. Infectious diseases other than tuberculosis are not important causes of death today. The scene is changing and social medicine must now concern itself increasingly with other forms of preventive medicine such as cancer of the lung, coronary thrombosis, rheumatism and mental deviations from the normal.

Indeed, there are few pathological conditions which have not their preventive aspects, and it is most useful and stimulating to witness what is happening overseas and to attempt to fit into South African conditions observations of value gained in this way.

MINUTES OF THE ANNUAL GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA

Following are the minutes of the Annual General Meeting of the Medical Association of South Africa, held in the B.E.S.L. Hall, Old Fort Road, Durban, on Wednesday, 11 September 1957, at 9.45 a.m.:

The President, Dr. J. S. du Toit, presided, and 53 other members were present. Ten proxies were received.

The President declared the Meeting duly constituted and stated that the notice convening the Meeting had been published in the *Journal* of 3 August 1957. This was taken as read, and the President welcomed members.

1. *Minutes* of the last Annual General Meeting held in Cape Town on 3 October 1956, which had been published in the *Journal*

of 27 October 1956, were taken as read, *Confirmed* and signed by the President.

2. *Annual Report and Balance Sheet*, published in the *Journal* of 27 July 1957: It was proposed by the Honorary Treasurer (Mr. J. D. Joubert), seconded by Mr. T. B. McMurray and *Resolved* that the Annual Report and Balance Sheet be adopted.

3. *Election of Auditors:* It was proposed by Dr. A. W. S. Sichel, seconded by Mr. Joubert and *Resolved* that Messrs. Gurney, Notcutt & Fisher, of Cape Town, be re-appointed Auditors for the year 1958, at a remuneration of £200 per annum.

4. *Induction of President:* Dr. du Toit paid tribute to Dr. H. Grant-Whyte, who was to succeed him in office, for the work done by him for the Association and the profession, and said that Dr. Grant-Whyte's election to the Presidency was a well-deserved honour. He then installed Dr. Grant-Whyte as President of the Association and wished him all success during his term of office. *Acclamation.*

Dr. Grant-Whyte then took the Chair and expressed his appreciation of Dr. du Toit's kind remarks. He said that he was grateful to the Association and to his Branch for the high honour which had been bestowed on him, and that he would carry out his duties in the Association to the best of his ability and endeavour to follow the example set by his predecessors. *Acclamation.*

5. *Thanks to Retiring President:* Dr. Sichel paid tribute to Dr. du Toit for the valuable service which he had rendered to the Association. He spoke of his long friendship with Dr. du Toit which dated back to their student days in Edinburgh, and said that on their return to South Africa they had both entered into the Association's service and had served together in various capacities ever since. Dr. du Toit had recently retired from the position of Honorary Treasurer which he had held for 30 years, and he himself would be retiring as Chairman of Federal Council, but they would continue to serve the Association as diligently as possible.

He then asked members to join him in expressing appreciation of the wonderful example set by Dr. du Toit and gratitude for his devoted service to the Association.

Members rose to acclaim Dr. du Toit.

Dr. du Toit expressed his thanks to the members.

The President then declared the Meeting adjourned until 8.30 p.m. on 16 September 1957, when the Adjourned Annual General Meeting was to take place in conjunction with the Opening Ceremony of Congress.

ADJOURNED ANNUAL GENERAL MEETING OF THE MEDICAL ASSOCIATION OF SOUTH AFRICA, AND OPENING CEREMONY OF THE 41ST S.A. MEDICAL CONGRESS, HELD IN THE CITY HALL, DURBAN, ON MONDAY, 16 SEPTEMBER 1957, AT 8.30 P.M.

Platform Party: Dr. B. Crowhurst Archer, Prof. the Rev. Dr. R. Craig, Dr. N. A. Rossiter, Prof. Guy Elliott, Mrs. H. Grant-Whyte, Dr. A. W. S. Sichel, Dr. J. S. du Toit, Dr. J. H. Struthers, Mr. D. G. Shepstone, Dr. H. Grant-Whyte, The Mayor of Durban, Dr. R. Schaffer, Dr. E. W. Turton, Mr. J. D. Joubert, Dr. T. C. Routley, Dr. A. Broomberg, Dr. A. H. Tonkin, Dr. K. W. Dyer, Dr. S. Disler.

1. *Invocation:* Prayer was offered by the Rev. Dr. R. Craig.

2. *Opening of Congress by Administrator of Natal:* Mr. Shepstone said that both as Chancellor of the University of Natal and Administrator of the Province, he wished to welcome to the country and particularly to Durban the members of the Medical Association and their many distinguished guests, some of whom had travelled thousands of miles to be at the Congress. He extended a special welcome to Prof. Guy Elliott who was attending as the First President of the newly-formed College of Physicians and Surgeons of South Africa, and to Sir Russell Brain, Sir Harry Platt and Mr. Joseph Wrigley, who had come to the country to be present at the first Fellowship examinations of the College which were to be held in Cape Town immediately after Congress. He congratulated the President and Council of the College on their achievements in so short a period of time.

Mr. Shepstone stated that the Governor General was unable to be present that evening on account of illness developed during his overseas tour. On behalf of Congress he expressed deep regret at his unavoidable absence and extended good wishes for a speedy recovery.

He said that the Medical Association of South Africa was the parent body, having been formed in 1927. He spoke of the objects of the Association and said he considered that these had been achieved over the last 30 years by holding regular meetings of the numerous local Branches and Specialist Groups and also by holding an Annual General Meeting and a Congress in every second year.

The Natal Coastal Branch was deeply sensitive of the honour of acting as host to the Medical Association on this occasion. He spoke of Durban's reputation for its hospitality. He mentioned that this Congress was more international in character than any previous Congress. He referred to the number of distinguished people present who had come from overseas to take part.

His Honour stated that many of the scientific papers to be presented at this gathering would make public for the first time recent advances in the various fields of medical science. He spoke of the rise of modern medicine and how it had removed much of the fear of illness and old age. Modern medicine had not completely dispelled fear, but at least it had rationalised it to the extent of producing a better pre-operative condition for the patient and a greater chance of recovery. The complete conversion of the Native population to the efficacy of the White man's medicine had still to be realised. Today our hospitals were flooded out; from the remotest parts of Zululand the Zulus and their wives came to reach the White man's medicine and marvels. Medical science had changed the human lot with great benefit. The search for knowledge and truth remained unabated.

In declaring the Congress open, Mr. Shepstone said that he did so with pride and pleasure, knowing full well its importance and significance to the development of medical science in South Africa. *Acclamation.*

3. *Presentations:*

(*a*) *Insignia of Immediate Past President:* The Secretary presented Dr. J. S. du Toit to the President and asked him to present him with a miniature of the insignia of office. This was done amid *Acclamation.*

(*b*) *Insignia of Past Chairman of Federal Council:* The Secretary presented Dr. A. W. S. Sichel and asked the President to present to him a miniature of the badge of office. This was done amid *Acclamation.*

(*c*) *Badge of Office of President's Lady:* The Immediate Past President presented to Mrs. Grant-Whyte the insignia of President's Lady, amid *Acclamation.* Dr. du Toit conveyed Mrs. du Toit's regret at her inability to be present, and her good wishes to Mrs. Grant-Whyte for a pleasant term of office. *Acclamation.*

4. *Presentation of Awards:*

(*a*) *Bronze Medals:* The Secretary read citations in the names of Mr. B. A. Armitage, Dr. A. Broomberg, Dr. C. M. Grundlingh, Dr. M. Shapiro and Dr. R. Theron, and presented these members to the President for the award of the Bronze Medal. The Medals were presented amid *Acclamation.*

(*b*) *Hamilton-Maynard Memorial Medal for* 1956: The Secretary announced that the Hamilton-Maynard Memorial Medal for 1956 had been awarded to Dr. Geoffrey Dean, of Port Elizabeth, for his paper entitled 'Porphyria, a Familial Disease: Its Diagnosis and Treatment', published in the *S.A. Medical Journal* of 21 April 1956. The award was made in absentia, amid *Acclamation.*

(*c*) *Leipoldt Memorial Medal for* 1956: The Secretary announced that the Leipoldt Memorial Medal for 1956 had been awarded to Dr. H. Braude, of Kroonstad, for his paper entitled 'Phenylketonuria', published in the *S.A. Medical Journal* of 28 January 1956. The award was made in absentia, amid *Acclamation.*

5. *Presidential Address* Dr. Grant-Whyte then delivered his Address.[1] This was received with *Acclamation.*

At the close of the Ceremony, a Presidential Reception was given at the Marine Hotel.

1. Grant-Whyte, H. (1957): S. Afr. Med. J., **31**, 945.

UNIVERSITEIT VAN PRETORIA

UITSLAE: M.B., Ch.B.: NOVEMBER 1957

Volgens berig van die eksaminatore en onderhewig aan bekragtiging van die Fakulteitsraad, het die volgende studente die finale eksamens wat in November 1957 gehou is, geslaag:

Bekker, J. H.
Bonnet, W. J. B.
Botha, G. J. B.
Crous, J. J.
de Beer, J. J. C.
de Beer, L.[1]
de Swardt, H. F.
de Villiers, J. F.
de Villiers, P. K.
Erasmus, J. A.
Faurie, J. P.
Graham, A. F.
Hamersma, T.
Jansen, L.[2]
Kachelhoffer, R. J. F.
Kallmeyer, J. C.[3]
Keevy, C. M.
Lindeque, W. J. M. P.
Lombard, N. M.
Maree, J. S.
McGill, D. J.[3]
Naude, M. C.
Nel, G. C.
Nel, P. J. C.
Opperman, J. M.[4]
Pretorius, H. A.
Pretorius, J. J. S.
Reisner, S. H.
Schoeman, P. F. C.
Schoeman, S. J.
Schoonees, J. A.
Serfontein, J. H.
Siegrühn, G. C.
Snyman, J. D.
Steyn, J. C.
v. d. Merwe, S. W.
v. Rensburg, W. H. J.
v. Rooyen, J. C.[3]
Venter, J. L.
Visser, E. J.
Zöllner, W. B.*

1. Onderskeiding in Obstetrie en Ginekologie (te Groen medalje).
2. Onderskeiding in Radiologie.
3. Onderskeiding in Kindergeneeskunde.
4. Onderskeiding in Interne Geneeskunde.

* Onderhewig aan die voorlegging van 'n sertifikaat van volle vrystelling van die G.M.R.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

'V-Cillin K' (Penicillin V Potassium, Lilly)

The Eli Lilly International Corporation announces 'V-Cillin K'—a new oral penicillin—and supplies the following information:

'V-Cillin K' combines the acid-stability of penicillin V with immediate solubility. Although Penicillin V is stable in gastric acid, not all of it becomes soluble within the time necessary for it to pass through the duodenum. 'V-Cillin K', however, is not only resistant to gastric destruction but is also readily soluble at gastric and duodenal pH. 'V-Cillin K' becomes completely soluble at pH 6-8; therefore, theoretically, *all* of the 'V-Cillin K' is available for absorption into the blood stream from the duodenum (considered to be the major site of penicillin absorption) and provides immediate high blood levels. The major advantages of 'V-Cillin K' are:

1. *Higher, faster blood levels*

Following the administration of 250 mg. of 'V-Cillin K', high blood levels (5·04 units per ml.) appear within 15 minutes: peak levels in the range of 6-8 units per ml. occur within 30 minutes. Levels reached in the first 2 hours are at least 4 times those from comparable doses of penicillin G. These levels are also achieved faster than from comparable intramuscular doses of soluble penicillin G potassium.

2. *Greater total penicillaemia*

'V-Cillin K' provides more penicillin in the blood for combating infection than do comparable doses of oral penicillin G, penicillin V acid and calcium penicillin V. (Definitive studies are now being conducted to establish the relative blood levels and penicillaemias obtained from 'V-Cillin K' and intramuscular procaine penicillin G.)

3. *Parenteral penicillin response*

The immediate high blood levels from 'V-Cillin K' simulate those obtained with injectable forms; therefore it is safe to assume that 'V-Cillin K' may be used satisfactorily to treat most infections formerly treated with parenteral penicillin. This is a major advantage, for both penicillin V and 'V-Cillin K'.

Blood levels in the range of 6-8 units per ml. are now attainable with an oral penicillin dosage of 250 mg. 'V-Cillin K' promises to be a significant contribution to the armamentarium of the practising physician, and is therefore a major improvement in oral penicillin.

Penicillin blood levels following the administration of equal doses of penicillin V and 'V-Cillin K' are almost identical *after* the second hour. The major advantage of 'V-Cillin K' is the extremely high blood level obtained in the first 2 hours following administration. This advantage can only be attained through the use of uncoated tablets which are specially designed to disintegrate very rapidly following ingestion.

Production Information. 'V-Cillin K' is available as:
Tablets No. 1830, 'V-Cillin K', 125 mg., in bottles of 10 and 50.
Tablets No. 1831, 'V-Cillin K', 250 mg., in bottles of 10.
(at the same prices as Penicillin V, Lilly, Pulvules.)

* * *

Steclin-V (Squibb Tetracycline Posphate Complex)

Squibb Laboratories (Pty.) Ltd. supply the following information:

Steclin-V is the new phosphate complex of tetracycline, providing faster and higher initial blood levels for rapid, well tolerated and fully effective broad-spectrum antibiotic therapy. Each Steclin-V capsule contains tetracycline phosphate complex equivalent to 250 mg. of tetracycline hydrochloride.

Action

Steclin-V has pronounced anti-microbial activity against a wide range of pathogenic organisms, including gram-positive and gram-negative bacteria, spirochetes, certain large viruses, certain rickettsias and *Endamoeba histolytica.*

Advantages

Steclin-V produces faster and higher initial antibiotic blood levels than are attainable with older forms of tetracycline. It is readily absorbed from the gastro-intestinal tract and diffuses rapidly into body tissues to combat infection. Steclin-V is especially well tolerated and causes a minimum of irritation in the intestine. Since it contains no free sodium, Steclin-V is not contraindicated for the patient whose sodium intake must be restricted.

Indications

Steclin-V is indicated for the many common infections, including those of the respiratory, gastro-intestinal and genito-urinary systems, which are amenable to tetracycline therapy. Infections caused by gram-positive and gram-negative bacteria, spirochetes, certain large viruses, certain rickettsias and *Endamoeba histolytica* can be expected to respond. Because of its wide range of anti-microbial activity Steclin-V is particularly useful in the treatment of mixed infections with susceptible organisms.

Dosage

The suggested minimum adult dosage is one capsule 4 times daily. Higher dosages, such as 2 capsules 4 times daily, may

be required for severe infections or for those which do not respond to the smaller dose. The paediatric dosage should be 10-20 mg. per lb. of body-weight each day, in divided doses, according to the type and severity of the infection.

Treatment for all patients should generally continue for 24-48 hours after symptoms and fever subside. However, it is advisable to treat streptococcal infections for a full 10 days to prevent the development of rheumatic fever. Even more prolonged therapy is necessary for subacute bacterial endocarditis and may be required in certain staphylococcal infections.

Note. With the use of any broad spectrum antibiotic the patient should be carefully watched for signs of secondary infection caused by non-susceptible organisms, particularly *Candida Albicans* (*Monilia*). If such infections appear Steclin-V should be discontinued and other appropriate measures taken. Moniliasis may be treated or prevented with Mycostatin (Squibb Nystatin).

Supply: Steclin-V capsules, each containing tetracycline phosphate complex equivalent to 250 mg. of tetracycline hydrochloride, are supplied in bottles of 16 and 100.

PASSING EVENTS : IN DIE VERBYGAAN

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town, as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria.

Failure to advise the Association can only result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as to those who change their addresses within the Union.

* * *

New Journal—Diseases of the Rectum and Colon. J. B. Lippincott Company of Philadelphia are publishing this new journal on behalf of the American Proctologic Society and the new journal will be handled in this country by Pitman Medical Publishing Co. Ltd., 45 New Oxford Street, London, W.C. 1. The Journal will start publication in January 1958. It will be published every two months and the subscription, including post, will be £5 per annum. It will be crown 4to size and each issue will contain at least 96 pages. The policy of the Journal will be to provide a centralized source of authoritative information covering the current advance in the medical and surgical treatment of diseases of the colon, rectum and anal canal. Contributions will be international, covering basic research in metabolism, pathology, virology, radiology, and the clinical practice of medicine and surgery, supplemented by comprehensive abstracts of world literature.

* * *

Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 15-21 November 1957:

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	14	5	–	–	19
Cape Province ..	3	2	3	–	8
Orange Free State..	–	2	–	–	2
Natal	–	–	–	–	–
Totals	17	9	3	–	29

Plague. Cape Province: One (1) fatal Native Case in the Uitenhage Divisional Council Area. Confirmed by laboratory tests. All necessary precautions taken.

Smallpox: Nil.

Typhus Fever. Cape Province: Four (4) Native cases in the Glen Grey District. Confirmed by laboratory tests. All necessary precautions taken.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following contributions to the Benevolent Fund during July, August and September 1957, are gratefully acknowledged.

Votive Cards in memory of:

Prof. M. van den Ende *by* Dr. A. Helfet and S.A. Paediatric Association.

Dr. A. M. Moll *by* Dr. and Mrs. A. W. Sichel, Dr. L. Mirvish, Dr. B. D. Knoblauch, Dr. A. I. Goldberg and Dr. P. W. J. Keet and Family.

Mr. A. Roup *by* Dr. and Mrs. M. Meyer.

Mrs. E. Basson *by* Miss C. L. Starke and Fiancé.

Baby Sophia Cilliers *by* Dr. C. J. Levy.

Mr. Justice Grindly-Ferris *by* Dr. and Mrs. A. J. v. d. Spuy.

Mrs. Woods, mother of Dr. J. D. Woods *by* Dr. F. E. Bamfort, Dr. M. T. S. Conradie, Dr. T. H. Whitsitt, Dr. N. Kemsley Pein, Dr. R. B. Peckham, Dr. S. J. Adendorff, Dr. C. C. Ackerman, Dr. C. G. Williams, Dr. N. A. A. v. Buuren, Dr. W. Fabian, Dr. H. A. Kalley, Dr. N. M. Thompson, Dr. P. Lewis, Dr. Borrowdale, Dr. R. R. McKenzie, Dr. D. H. Biggs, Dr. H. M. L. Lund, Dr. A. H. Baxter, Dr. J. D. M. Barton, Dr. R. L. Tibbit, Dr. K. Strauss and Dr. J. F. Rivers-Moore.

Dr. H. Q. F. Thompson *by* Dr. A. J. Orenstein.

Miss Florence Abraham *by* Dr. D. G. Cowie.

Dr. Percy A. Green *by* The Secretary and Staff, Witwatersrand Gold Mines Employees Provident Fund.

Dr. G. S. Andrews *by* M.A.S.A. Natal Coastal Branch, Dr. F. Walt, Dr. V. L. Asherson and Dr. P. G. Large.

Dr. G. v. R. Mostert *by* Prof. L. J. te Groen, Dr. H. Aneck-Hahn, Dr. D. A. Fowler Dr. S. Donen and Family, Dr. C. M. Grundlingh and Family and M.A.S.A. Northern Tvl. Branch.

Dr. S. Levy *by* Mr. J. A. Douglas.

Mrs. Kinkead *by* Mr. J. A. Douglas.

Mrs. R. Stewart *by* Mr. J. A. Douglas.

Mr. K. Newby *by* Mr. J. A. Douglas.

Dr. O. Popper *by* The Johannesburg Ear, Nose and Throat Group, Dr. S. Heymann and Dr. S. Javett.

Dr. E. Sthamer *by* Dr. H. Aneck-Hahn and Prof. L. J. te Groen.

Mr. G. Bacon *by* Dr. Bok, Dr. Ofsowitz, Dr. Botha and Dr. Goldblatt.

Mr. F. W. L. Liesching *by* Dr. E. R. Hafner.

Dr. R. J. Chadwick *by* Dr. H. Aneck-Hahn.

Mrs. M. E. Fuller *by* Dr. M. H. Judd.

Total amounts received from Votive Cards: £74 6s. 0d.

Services Rendered to:

Mrs. B. Kavalsky *by* Dr. W. P. Mulligan.

Late Miss E. Beyers *by* Dr. Ziady, Dr. Schulenberg and the Staff of the Volkshospitaal, Pretoria.

Dr. Hugo Hallatt *by* Mr. N. Louw, Dr. P. E. Dreyer, Dr. J. G. W. Sutherland and Dr. W. Greeff.

Lindy de Wet *by* Dr. M. Levin and Dr. B. Bellon.

Dr. J. S. du T. de Wet *by* Mr. S. Eisenhammer.

Mrs. F. de C. Keogh *by* Dr. L. J. A. Lowenthal.

Robert, son of Dr. O. M. Haarburger *by* Dr. Heselson, Dr. Abelsohn and Dr. Cullis.

Various members of the family of Dr. A. Goldberg *by* Dr.

Mannie Stein, Dr. F. J. Davidson, Dr. Oscar Schulman, Dr. D. F. Standing, Dr. Harry Curwen and Dr. N. Shapiro.

Mrs. McCartney *by* Dr. G. Piesold and Dr. J. Coster.

Mrs. J. Friedman *by* Dr. F. P. Reid, Dr. A. J. Tinker and Dr. K. G. Irving.

Dr. L. Ruskin *by* a Colleague.

Ann, Daughter of Prof. and Mrs. B. Beinart *by* Dr. N. L. Murray.

E. Dunbar *by* Dr. D. A. Edington.

Dr. A. A. M. Coutts *by* Dr. J. Macfadyen and Dr. L. Mundy.

Dr. R. Forsyth *by* Dr. Clegg and Dr. Finlayson.

Mrs. I. B. Taylor *by* Dr. W. Weinberg.

Infant daughter of Dr. and Mrs. Taylor *by* Dr. Seymour Heyman.

Total amount received from Services Rendered: £131 16s. 6d.

Donations by:

	£	s.	d.
Cape Western Branch Collection Box	10	5	7
Dr. P. Conradie and Dr. L. M. van Schalkwyk ..	5	0	0
Proceeds from Alan Sichel Golf Competition ..	10	0	0
Cape Western Branch Collection Box	7	3	10

Total amount received from Donations: £32 9s. 5d.

Grand Total: £238 11s. 11d.

REVIEWS OF BOOKS : BOEKRESENSIES

THE BRITISH ENCYCLOPAEDIA OF MEDICAL PRACTICE

The British Encyclopaedia of Medical Practice—Pharmacopoeia 1957. Second Edition. Pp. viii + 32. £3 7s. 6d., postage 2s. extra. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

Contents: Publishers' Announcement. Table of Weights and Measures. Pharmacopoeia. Discontinued Products. Manufacturers' Addresses. Condition. Index.

This new and enlarged edition will be welcomed by the medical profession and by pharmacists, especially those in hospital practice. The number of proprietary preparations offered to doctors is legion, and new ones are continually being presented, while a lesser number fade into obscurity. All who deal with drugs know how difficult it is to keep pace with these changes. The publishers of the British Encyclopaedia of Medical Practice have performed a great service in making this Pharmacopoeia available. More than 1,400 products are described, as against 500 in the first edition. A certain amount of repetition does obviously occur, since brand names for the same product may differ; however the descriptions vary so that much general information is provided and the book is virtually a textbook of pharmacology. The publishers do not advise which products should be used, but leave the decision regarding suitability to the reader. The preparations are arranged in alphabetical order, and for each there is a description of the drug, pharmacology, indications and contraindications, side effects or toxicity, dosage, method of administration and presentation (packings). A list of drugs which have been discontinued since the first edition was published is given separately. A 'condition index' (32 pages greatly facilitates reference to the agents which can be used in connection with various diseases. N.S.

ATLAS OF BLOOD AND MARROW DISEASES

Blood and Bone Marrow Patterns. By G. D. Talbott, M.D., and Elmer R. Hunsicker, B.S. and Jonah Li, M.D. Pp. 59. 140 Colour Plates. $12·00. New York and London: Grune & Stratton, Inc. 1957.

Contents: Preface. Cell Morphology. Rubricytic Series. Myelocytic Series. Monocytic Series. Megakaryocytic Series. Plasmacytic Series. Lymphocytic Series. *Cell Patterns.* Anemias. Leukemias and Lymphomas. Unrelated Abnormalities.

This publication comprises a comprehensive atlas of coloured photo-micrographs of peripheral blood smears and bone marrow aspirates found in all the important blood diseases.

In the preface the authors emphasize the importance of cell patterns, rather than individual cell morphology and stress that the textual content of the book has been kept to a minimum. In the reviewer's opinion this publication would benefit by additional textual information and annotation.

Morphological terminology varies in different parts of the English-speaking world and a graphic table of the terminology used would be helpful at the outset. Quite frequently one is not certain whether the illustration is of peripheral blood or of bone marrow aspirate. Some indication of the type of staining used would be welcome. These points may be obvious to experienced haematologists but are not so clear to students and relatively inexperienced practitioners. It is by such people that this book would chiefly be used.

The reproductions of the photo-micrographs are excellent and the book is cleverly bound with a ring fold mechanism, which allows the pages to lie completely flat.

It is probably extremely difficult to strike the happy medium in an atlas such as this, but there is little doubt that more in the way of explanation and textual information would make this publication much more understandable to the casual haematologist or student of medicine.

M.J.B.

ELLIS'S ANATOMY

Ellis's Anatomy. Second Edition. Revised and edited by J. A. Keen, M.B. (Lond.), F.R.C.S. (Eng.). Pp. viii + 470. Illustrated. 42s. net. Pietermaritzburg: University of Natal Press. 1957.

Contents: Introduction. *Section I.* Upper Limb. *Section II.* Lower Limb. *Section III.* Abdomen, Perineum and Pelvis. *Section IV.* Head and Neck. *Section V.* Thorax. *Section VI.* The Brain. Index.

The first edition of this manual of practical Anatomy was produced in 1946, when Professor Keen was still Senior Lecturer in Anatomy in the University of Cape Town. Although based upon G. V. Ellis's classic 'Demonstrations of Anatomy', it was so thorough a revision as to constitute in effect a new text built around the old illustrations. Professor Keen has now found time, among his responsibilities as head of the Department of Anatomy in the Durban Medical School, to bring out a new edition, which has been handsomely produced by the University of Natal Press. In addition to some new illustrations and improvements to a number of the older ones, the text of this edition has been carefully and critically revised. In this task Professor Keen has been assisted by some of his colleagues in Durban and also by his son Dr. E. N. Keen, now Senior Lecturer in his father's room in Cape Town.

With the constantly rising cost of books, the advantage to the student's pocket of a single-volume manual of regional Anatomy is more evident in 1957 than it was in 1946. To produce a comprehensive text within this compass is a very considerable feat, which Professor Keen has most ably accomplished. His text, however, has merits beyond those of conciseness allied to accuracy. Not least of these, to the practical teacher, is the carefully thought-out plan of dissection upon which the work is based. This aims at preserving as much as possible of the structure of the body intact to the end of the dissection programme. Not only is this advantageous to teaching, but it imposes a discipline on the student which cannot but be of value, more especially to the aspirant surgeon.

While Professor Keen has, as he states, had in mind a particular sequence of dissection which he regards as most satisfactory, the reviewer's experience in two medical schools has been that this text can equally well be used as a guide by students following quite different programmes.

Many teachers of Anatomy will confess their abiding affection for the manual which they themselves used as students. Another group will find all existing manuals wanting; everyone who belongs to this group will in the end be driven, as Professor Keen has been, to produce his own manual. Having had the experience of either learning or teaching from six manuals of the most diverse character, this reviewer is most grateful to Professor Keen for his meritorious

attempt to realise on earth the ideal manual whose pattern we all firmly believe to be laid up in Heaven.

L.H.W.

ANATOMY AND SURGERY OF THE LIVER

Le Foie Études Anatomiques et Chirurgicales. Par C. Couinaud. Pp. 530, avec 256 figures, 1 planche en couleurs (17 x 25). 4·500 fr. Paris: Masson et Cie. 1957.

Table des Matières: Livre I. Études Anatomiques. Livre II. A La Recherche De Techniques Chirurgicales. Addendum. Bibliographie. Table des Figures. Table des Matières.

A Frenchman's thoughts on the liver are always profound; even the layman greets one with enquiries about the state of one's liver. This lay concern in France is reflected by the assiduous researches of her medical scientists in this subject.

The delight a Frenchman takes in his food and in women is balanced by the melancholy about his liver, the former two may be contributing factors explaining the third sentiment. This book, with French text, is the result of 6 years of research at the anatomy laboratories of the Faculty of Medicine in Paris.

The anatomy of the hepatic ducts and vessels has been investigated by techniques using an injectable solution of polyvinyl which hardens in the vessels and biliary ducts. This is followed by corrosion of the liver parenchyma, leaving the exposed vessels. The anatomical distribution of these vessels has been verified by dissection of fresh specimens and by series of sections.

This work is divided into 2 sections: Book I deals with the anatomy and includes:—

Ch. I. General scheme of vascular and biliary circulation.
Ch. II. Segmentation of Liver architecture and surgical anatomy.
Ch. III. Embryology.
Ch. IV. Comparative anatomy—from lower vertebrates to reptiles, birds and mammals.
Ch. V. The hepatic pedicle.
Ch. VI. The sub-hepatic pedicle.
Ch. VII. The vasculo-biliary envelopes of the Liver.
Ch. VIII. The variations and anomalies of the gross anatomy of the Liver.
Ch. IX. Nomenclature Bibliography.

No trouble has been spared in comparing the findings of researchers in other countries who have done similar work.

Book II deals with the researches in technique of liver surgery from pre-operative examination, types of incisions, techniques of excision of segments of varying degree of magnitude, with both abnormal and normal anatomical features, to drainage of the biliary canals and peritoneum.

A further two sections deal with the surgery of the biliary system, and carcinoma of the gall bladder from the aspect of surgical technique.

This book is profusely illustrated with diagrams and a few photographs, both of which are a great asset in clarifying the detailed descriptions in the text.

In summary, this work would be of great interest to the anatomist and to the surgeon specializing in liver surgery.

R.B.

THE FIRST FIVE YEARS

The Normal Child—Some Problems of the First Five Years and Their Treatment. Second Edition. By Ronald S. Illingworth, M.D., F.R.C.P., D.P.H., D.C.H. Pp. xii + 356. 68 Figures. 33s. London: J. & A. Churchill Ltd. 1957.

Contents: Section 1. *Feeding Problems.* 1. Breast Feeding or Artificial Feeding? 2. The Feeding Schedule. 3. Some Breast Feeding Problems. 4. Breast and Nipple Difficulties. 5. Insufficiency of Milk. 6. Difficulties in the Breast-Fed Baby. 7. The Artificially Fed Baby. *Section* 2. *Physical Problems.* 8. The Assessment of Physical Development. 9. The Skull. 10. The Teeth. 11. The Skin and Umbilicus. 12. The Breasts and Genitals. 13. Some Miscellaneous Problems. 14. The Prevention of Infection. *Section* 3. *Developmental Problems.* Introduction. 15. Is Prediction Possible? 16. The Normal Course of Development. 17. General Factors which affect the Course of Development. 18. Retardation in Single Fields of Development and Factors Responsible. 19. Developmental Diagnosis. *Section* 4. *Behaviour Problems.* Introduction. 20. Relevant Features of the Psychological Development of the Child. 21. Parental Attitudes and Management. 22. Discipline and Punishment. 23. Anorexia and other Eating Problems. 24. Sleep Problems. 25. Problems of Sphincter Control. 26. Crying. Temper Tantrums. Breath-holding Attacks. 27. Body Manipulations. 28. Jealousy. Fears. Shyness and other Problems. 29. The Prevention of Accidents. 30. Toys and Play. Nursery School. 31. The Sick Child. The Child in Hospital. References. Additional Reading Recommended. Index.

The fact that this book has now achieved a second edition, after the appearance only three years ago of its reprinted first edition, is indicative of its value. The author has wisely extended the scope of his subject by 2 years to include the first 5 years of life; this is indicated by the sub-title of the book, which incidentally, is more descriptive of the actual content of this work than its title. It has always been difficult, if at all possible, to define normality but this book achieves much in assessing the widest aspects of normal happenings in the infant and young child.

The chapter dealing with accident prevention is extremely useful and this aspect cannot be sufficiently stressed by all concerned with child care. No less important in the contents is the discussion on the hospitalized child in the last chapter of the book.

The works of Dr. Arnold Gesell influence the chapters dealing with the development of the young child and these are acknowledged extensively in this book.

This edition has much fresh material and also lists numerous useful recent references, together with additional recommended reading matter. It is well-bound, of manageable size, printed on fairly good paper and has a useful index. The book is a highly recommended readable volume for all concerned with child care in its widest sense.

I.K.

TUBERCULOSIS NURSING

Tuberculosis Nursing. Second Edition. By Jessie G. Eyre, M.A., S.R.N., B.T.A. (Hons.). Pp. xvi + 354. Illustrations 98. 25s. net. London: H. K. Lewis & Co., Ltd. 1957.

Contents: Section I. What is Tuberculosis? *Section II.* The Course and Clinical Picture of Pulmonary Tuberculosis. *Section III.* Sensitivity and Immunity. *Section IV.* Tuberculosis in Association with Pregnancy—In Mental Hospitals —Tuberculosis in Industry. *Section V.* Diagnostic Procedures. *Section VI.* General Principles of Treatment of Pulmonary Tuberculosis. *Section VII.* General Nursing Care in the Sanatorium. *Section VIII.* The Feeding of the Tuberculous Patient. *Section IX.* Ward Hygiene: Prevention of Spread of Tuberculosis and other Infections in the Sanatorium. *Section X.* Special Nursing Procedures. *Section XI.* Chemotherapy in the Treatment of Tuberculosis. *Section XII.* The Administration of Medicines and Drugs. *Section XIII.* Collapse Therapy. *Section XIV.* Thoracoscopy and Adhesion Section. *Section XV.* Chest Surgery in the Treatment of Tuberculosis. *Section XVI.* Complications of Pulmonary Tuberculosis (1). *Section XVII.* Complications of Pulmonary Tuberculosis (2). *Section XVIII.* Tuberculous Arthritis. *Section XIX.* The Dying Patient. *Section XX.* Tuberculosis Care and After-Care Schemes. Appendices. Index.

This book has been written for those nurses who intend to specialize in tuberculosis nursing. The layout is pleasing and the illustrations, appendices and index are clearly laid out.

The scope of the subject matter is broad and the author has dealt with the complexities of the nursing problems quite adequately. There is, however, one exception to this in the chapter dealing with chest surgery. The remarks on post-operative care are on the brief side and could well be amplified, particularly in a book for the intending or practising specialist nurse.

The impression gained on reading this chapter is, that the post-operative period is a routine stage of recovery without any particular problems of its own—a section on the recognition of anoxia would not be out of place here.

However, in spite of this omission it can be recommended as a reference book for those nurses who intend to specialise in this work.

J.B.P.

ANATOMICAL NOMENCLATURE

Nomina Anatomica. Von Prof. Dr. Fr. Kopsch. 5 Auflage. Eine vergleichende Ubersicht der Basler, Jenaer und Pariser Nomenklatur Bearbeit von Prof. Dr. K. N. Knese. xx + 147 Seiten. DM 6·40. Stuttgart: Georg Thieme Verlag. 1957.

The new (5th) edition of the late Professor Kopsch's "Nomina Anatomica", revised by Professor Knese of Kiel, sets out in parallel colums the Basle Nomina Anatomica (B.N.A.) of 1895, the Jena revised nomenclature (I.N.A.) of 1935, and that adopted at the Sixth International Congress of Anatomists in Paris in 1955 (P.N.A.), indicating by symbols the terms in the Paris list which differ either from those of the B.N.A. or those of the I.N.A.

This tabulation marks a new stage in the process of securing an internationally accepted anatomical terminology. Before

1895 there was no generally accepted system of nomenclature, though the anatomists of each country tended to adhere to a common usage. The 'Old Terminology' dear to a generation of British anatomists now rapidly vanishing was of this sort. The Basle terminology was formulated largely by German anatomists, with some help from those of other countries; it therefore followed current German practice.

By 1920 the B.N.A. was used in most European countries and in the U.S.A. British anatomists had been slow to come into line, and it was never regularly accepted in France. By this time also it was widely felt that the B.N.A. could be improved upon. In 1933 the Anatomical Society of Great Britain and Ireland adopted a modified system, the Birmingham Revision (B.R.); this was intended to be put forward as a basis for international agreement. At the same time, German anatomists developed the Jena terminology, which modified the B.N.A. much more radically in certain directions, especially towards bringing the language of human anatomy into line with that of vertebrate comparative anatomy. A move towards an internationally agreed terminology was initiated at the Fourth International Congress of Anatomists in Milan in 1936, but was brought to nothing by the outbreak of war in 1939.

The climate of opinion after the war was much more favourable to international agreement. At the Fifth International Congress of Anatomists, held in Oxford in 1950, a broadly representative international committee was set up, and the proposals of this committee took shape in the nomenclature adopted at the Paris meeting. Thus a great step appears to have been taken towards a universally accepted nomenclature. This nomenclature remains fairly close to the B.N.A., incorporating some obvious improvements from either the Birmingham or the Jena lists, and others suggested by the common experience of anatomists. Like the B.N.A., the Paris nomenclature has been set out in Latin as being the most useful international language for scientific purposes. Its compilers have however been at pains to ensure that terms can be readily translated into modern languages. The importance of this will be clearly recognised in its relation to the development of anatomical terminology in a 'new' language such as Afrikaans.

In South Africa, as elsewhere, there will for a long time to come be teachers of Anatomy whose foundations will have been laid in either the B.N.A., the Birmingham Revisions, or the Jena nomenclature. The adjustment for any of these to the Paris nomenclature will be a less drastic process than that from the 'old' British terminology to the B.N.A. To those who have to make such an adjustment, this tabulation of the various nomenclatures will be of the greatest value.

L.H.W.

THE ELECTROCARDIOGRAM

The Electrocardiogram. Second Edition Revised. By Louis H. Sigler, M.D., F.A.C.P., F.C.C.P., F.A.C.C. Pp. xii + 312. Figs. 192. $8·75. New York and London: Grune & Stratton, Inc. 1957.

Contents: Preface to second edition. Preface to first edition. *Section I.* The Foundations of the Electrocardiogram. *Section II.* The Transmission and the Recording of the Heart Current. *Section III.* The Normal Electrocardiogram. *Section IV.* The Electrical Axis. *Section V.* Ventricular Gradient. *Section VI.* Mechanism of Unipolar Lead Deflections. *Section VII.* The Abnormal Electrocardiogram: Classification. *Section VIII.* Sinus Tachycardia, Bradycardia, Arrest, and Arrhythmia. *Section IX.* Premature Contractions, Extrasystoles, Ectopic Beats. *Section X.* Displacement of the Pacemaker. *Section XI.* Circus Movement Theory. *Section XII.* Paroxysmal Tachycardia: Tachycardia of Ectopic Origin. *Section XIII.* Auricular Flutter. *Section XIV.* Auricular Fibrillation. *Section XV.* Ventricular Fibrillation. *Section XVI.* Heart Block: Auriculoventricular. *Section XVII.* Bundle-Branch Block. *Section XVIII.* The Electrocardiogram in Cardiac Hypertrophy and Strain. *Section XIX.* Myocardial Infarction. *Section XX.* Myocardial Ischemia and Necrosis. *Section XXI.* Pericarditis and Myocarditis. *Section XXII.* Acute Cor Pulmonale. *Section XXIII.* Trauma and Strain Affecting the Heart. *Section XXIV.* The Electrocardiogram in Congenital Heart Disease. *Section XXV.* Electrocardiographic Changes in Various Constitutional and Toxic States. *Section XXVI.* Effect of Drugs and Chemicals on the Electrocardiogram.

Dr. Sigler's book deals very adequately with the important aspects of cardiography from the clinicians point of view. It is admirably illustrated from an experience which is obviously profound. The electrophysical basis of cardiography is explained more concisely and clearly than in most books on the subject and this helps to avoid the empirical approach, which is still far too common. The arrhythmias are dealt with particularly well, as are the changes in myocardial infarction.

Dr. Sigler devotes less than one page to the subject of auricular hypertrophy, which is a pity in view of our growing interest in this subject. The changes brought about by derangements in potassium metabolism should, I think, also have received more adequate attention. It is interesting that Dr. Sigler makes repeated reference to T-wave changes in the right precordial leads in healthy American Negroes. These, although not illustrated, are apparently similar to the ones which have been described by Grusin among Africans in this country.

All in all, I know of no better text book of cardiography, especially for those who need guidance in the interpretation of tracings in parts of South Africa away from the bigger centres.

S.J.F.

YEAR BOOK OF ENDOCRINOLOGY

The Year Book of Endocrinology. (1956-1957 Year Book Series). Edited by Gilbert S. Gordan, M.D., Ph.D., F.A.C.P. Pp. 377. $6.75. Chicago: Year Book Publishers, Inc. 1957.

Contents: Introduction. *Section* 1. The Pituitary Gland. *Section* 2. The Thyroid Gland. *Section* 3. The Parathyroid Glands, Calcium Metabolism and Metabolic Bone Diseases. *Section* 4. The Adrenal Glands. *Section* 5. The Reproductive System. *Section* 6. Carbohydrate Metabolism.

Year books are not really reviewable. It is plain that anyone interested in clinical endocrinology must possess this particular Year-book series. Nevertheless, as the editor (Dr. Gordan) admits, only a fraction of the publications on the subject have been reviewed. Incidentally an unusually high proportion of the articles mentioned were published outside the United States. Diabetes, under the head of 'Carbohydrate metabolism', being such a vast subject itself, is rendered scant justice. (A year book of Diabetic Science would not be out of place, one feels.)

In this volume there are 2 special, but over-brief, review articles—one by Elrick on glucagon and the other by E. L. Albright on the thyroid hormone. The editor's comments make up a fairly large portion of the book, and these, naturally, are largely personal opinion, but nevertheless they are usually helpful and sensible.

W.J.

PROGRESS IN PSYCHOTHERAPY

Progress in Psychotherapy—Volume 11. *Anxiety and Therapy.* Edited by Jules H. Masserman, M.D. and J. L. Moreno, M.D. Pp. viii + 264. $7.50. New York and London: Grune & Stratton Inc. 1957.

Contents: Preface. *Part I:* Introduction. *Part II:* Anxiety: The Background of Psychotherapy. *Part III:* Special Problems: Stresses and Techniques in Later Life. *Part IV:* Schools and Trends in Psychotherapy. *Part V:* Developments Abroad. *Part VI:* Summation. Author Index. Subject Index.

As long as those who are responsible for organising meetings of various medical groups continue to publish holus-bolus the proceedings of those meetings, the medical reading public will continue to suffer from literary indigestion. The present series of lectures have to some extent been edited and added to so that this volume is not quite such a patchwork as many other similar ventures, but the creaking, uneasy prose of many writers makes the going extremely heavy. Luckily some of the quicksands are to some extent marked. (In psycholese this might be put, 'Cued off in nominal frames of reference'.) Chapters headed 'Sociometric Milieu Therapy' and 'Biodynamic Therapy—an Integration' are their own danger signals, but one can never tell: 'Daseinanalysis' by Professor Boss of Switzerland brings a refreshing existentialist note into the forum, but a chapter invitingly labled 'Experimental Aspects of Anxiety' tells one a great deal about a large number of experiments but leaves the question of anxiety largely to the reader in his attempts to understand what it is all about.

Kurt Goldstein, although he does not say anything startlingly new is, as always, well worth while reading. Dr. Rollo May writes eruditely on 'Anxiety and Values' and uses plain, comprehensible English, and Dr. Cozin gives good practical advice on the running of Geriatric Units, but for the rest, one might say without being unjust, and not meaning to be unkind, that this is the sort of book which might be useful in a reference library but is not intended for armchair reading.

J.MacW.MacG.